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THE JOURNAL OF NEUROLOGY AND PSYCHOPATHOLOGY

Vol. XIII. JANUARY, 1933. No. 51.

Original Papers.

THE SOCALLED LAW OF ANTICIPATION IN MENTAL DISEASE.

By

ARTHUR S. PATERSON*, London.

The purpose of the present study is to examine and criticize a supposed law which has been given great prominence by some writers on the heredity of mental disease. It has been said that there is a tendency for an inherited disease to occur in the child at an earlier age than that at which it occurs in the parent. This may be expressed otherwise by saying that in the child the age of onset anticipates the age of onset in the parent. The law, which was postulated to account for the apparent earlier onset of the disease in the offspring, was therefore called the 'law of anticipation.' The mere fact of earlier onset in the child has also been called antedating, anteposition, and, in France, précession.

DEVELOPMENT OF THE CONCEPTION OF EARLIER ONSET IN THE OFFSPRING AS EXPRESSING A NATURAL LAW.

Observations on this subject were originally made outside the sphere of mental disease. Darwin[1] himself stated in his *Animals and Plants under Domestication* that there existed a strong tendency for disease to appear in the offspring at the same period of life as in the parent, but where this rule failed, the disease could occur at an earlier age than in the parent, whereas it was exceptional for the opposite to occur.

The French psychiatrist Morel[6], in his book *Traité des dégénérescences de l'espèce humaine*, was the first to lay great stress on the age of onset of mental disease in successive generations. He believed that there was to be found in some pedigrees what he called a 'progressive degeneration' in virtue of which the affected stock became extinct in the course of a few generations. He believed that the degeneracy in the first generation took the form of mild nervous disorders which appeared late in life. The

* Commonwealth Research Fellow, Maudsley Hospital.

predisposition to disease became more accentuated in the second and third generation, appearing at an earlier age and in a more intense form, until the members of the degenerate stock, being unable to propagate further, became finally extinct.*

It will be seen then that Morel believed that this ' progressive degeneration ' was of very frequent occurrence, even the rule, in mental disease.

Henry Maudsley[3], treating of the same subject, wrote: ' There appears to be at work a silent tendency in Nature to restore an insane stock to a sound type, if regeneration be possible, or to end itself, if degeneration is too bad to mend.' It will be noticed that this writer does not say that insane stocks die out by progressive degeneration, but merely that in many cases insanity goes hand in hand with sterility.

An English writer who examined particularly the age of onset of inherited disease was Nettleship, who did valuable work in collecting pedigrees relating to diseases of the eye. In a paper published with E. J. Lidbetter in *Brain*, 1913, he[4] described antedating as occurring in a stock whose members suffered from a complicated eye disease and different forms of mental disease. Nettleship stressed this apparent earlier onset in successive generations and believed it to be the expression of a natural law. He also referred to the fact that inherited disease appeared to occur at an earlier age in younger siblings than in older siblings. This he called ' fraternal anticipation,' a phenomenon which will be discussed later in this paper.

At Nettleship's instigation, the late Sir Frederick Mott started an enquiry into the inheritance of mental disease with a view to ascertaining whether it was the rule for mental disease to occur at an earlier age and in a more intense form in successive generations. If this were found to be so, then it might appear that there was a ' law of anticipation ' in virtue of which Nature finally brought a tainted stock to an end in the manner described. Mott accordingly instituted a card index system, in which the name of each patient in the London County Council Mental Hospitals, who had either a parent, child or other relative in one of these hospitals, was

* *Traité des dégénérescences de l'espèce humaine*, Paris, 1857, p. 565. ' Les médecins aliénistes . . . savent qu'un simple état névropathique des parents peut créer chez les enfants une disposition organique qui se résume dans la manie et la mélancholie, affections nerveuses qui, à leur tour, peuvent faire naître des états dégénératifs plus graves et se résumer dans l'idiotie ou l'imbécillité de ceux qui forment les derniers anneaux de la chaîne des transmissions héréditaires. J'ai constamment observé, pour ma part, que les enfants d'un père ou d'une mére aliénés présentaient, dès l'âge le plus tendre, des anomalies du côté des fonctions nerveuses qui étaient les signes les plus certains d'une dégénérescence ultérieure, lorsque rien n'était fait pour combattre un danger aussi redoutable. Le peril est plus imminent, et pour ainsi dire inevitable, lorsque l'herédité est double.'

recorded, together with the date of admission, age of onset, and type of psychosis of both relatives. Mott then compiled statistics which appeared to show that not only was the age at which the psychosis started much earlier in the children than in the parents, but also earlier in nephews and nieces than in uncles and aunts.

It is this work of Mott's which is especially considered in the present study. Other writers both in this and other countries have quoted their findings in support of the theory that tainted families become extinct in virtue of this hereditary law. It has seemed the best course, however, to direct the criticisms contained in this article against the work of Mott, because he was the most eminent English writer to support this theory, and was the first to give statistical backing to Morel's theory.

Mott[7] summed up his results in *Brain*, 1911. From his statistics one finds that insane children of insane parents appeared to become affected on the average some 24 years earlier than their parents. In view of this fact, Mott wrote: ' I noticed there was a general tendency for the insanity not to proceed beyond three generations. There is frequently a regression to normal type, or else the stock dies out. Not infrequently the stock dies out by the inborn tendency of insanity manifesting itself in the form of congenital imbecility, or the insanity of adolescence.' He[8] wrote further in the *British Medical Journal* that ' this signal tendency of insane offspring to suffer with a more intense form of the disease, and at an earlier age, is of great importance for the following reasons: first, it is one of nature's methods for ending or mending a degenerate stock; secondly, it is of importance to the physician, for he can say that there is a diminishing risk of the child becoming insane after he has passed twenty-five, a matter of great importance in the question of marriage; thirdly, it is of importance in connexion with the subject of social surgery of the insane, for when the first attack of insanity occurs in the parent, the children have for the most part all been born.'

SCOPE OF PRESENT ENQUIRY.

The material upon which Mott based these conclusions has continued to accumulate during the 20 years following 1911. The bulk of the material was taken from Colney Hatch, which is the largest London County Mental Hospital. Through the courtesy of the Superintendent, Dr. Brander, I have been able to go through over 4,000 cases, and compare the material which Mott used in 1911 with that which obtains at the present day. In what follows, then, I shall call attention to certain statistical fallacies which accounted for Mott's results. Some of these, indeed, were pointed out by Prof. K. Pearson[9] and Dr. D. Heron[2] soon after the publication of Mott's work. I shall try to show that much of the apparent anticipation was due to the fact that Mott's material ranged over a comparatively short period of time, a fact which, as we shall see, favours the appearance of a spurious antedating

in the children's generation. This is borne out by examination of cases during the 20 years following the publication of Mott's work. Finally, I shall attempt to show by analysis of Mott's own data that anticipation is only the rule where the parents become affected late in life.

EXAMINATION OF MOTT'S MATERIAL.

The accompanying diagram (fig. 1), reprinted by permission from *Biometrika*, Vol. X, 1914, p. 368; 'The Inheritance Factor in Insanity,' by D. Heron, embodies the data on which Mott chiefly relied to substantiate his

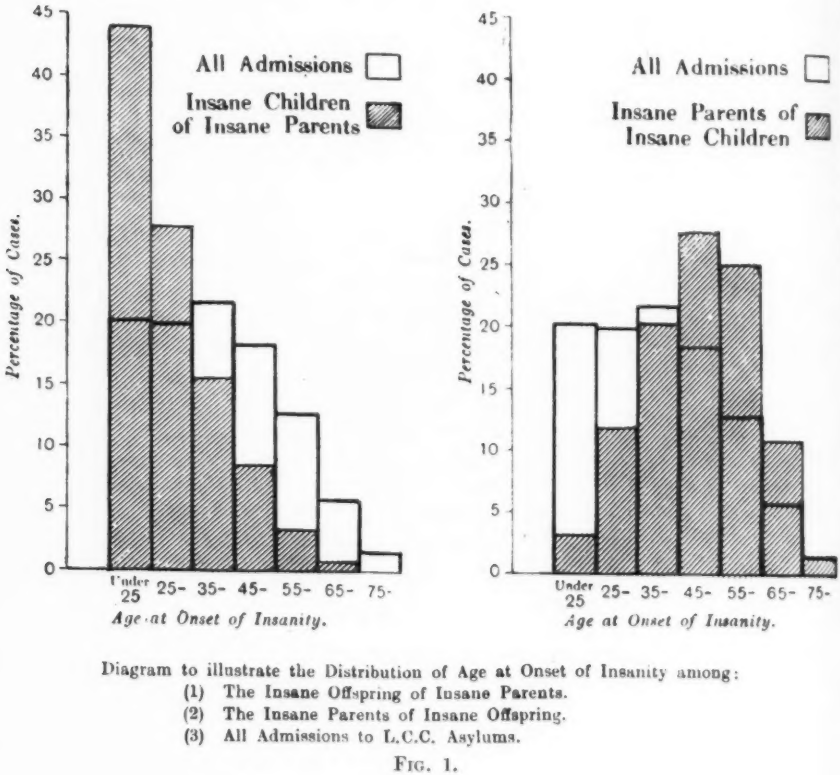

Diagram to illustrate the Distribution of Age at Onset of Insanity among:
(1) The Insane Offspring of Insane Parents.
(2) The Insane Parents of Insane Offspring.
(3) All Admissions to L.C.C. Asylums.

FIG. 1.

theory. The unshaded columns represent the percentage of all cases to be admitted at the various age periods. In the left-hand figure the shaded columns show the insane children of insane parents, and the percentage admitted at each period, while the shaded columns on the right indicate the same for insane parents of insane children.

If we consider first the right-hand figure, it is not difficult to understand why the parents do not constitute a fair sample of the general admissions, that is, why the parents' columns do not correspond to those of all admissions. The reason lies in the very fact of parenthood. If he is to be a parent at all, an individual must in all probability reach the age of 25 or 30 before falling a victim of mental disease. We therefore find very few parents affected before the age of 30. Since a relatively low percentage are first affected in early life, a relatively high percentage are affected in later life, and so there too the curve for parents, being much higher, differs from that for all cases admitted.

Mott at first compared the average age of onset of mental disease in the children with that in the parents, not with that in all cases admitted. Here then we see the first fallacy. He is comparing the children who may be affected from birth onwards with the parents who are only likely to be affected after becoming parents, that is, after the age of 25 or 30.

This fallacy can be best exemplified, as Professor Pearson pointed out, by considering cases of persons who have met with a violent death. In this case we should find that since about half of violent deaths in females occur before the age of 20, and about a quarter in males before that age, and since almost none of the present generation, in virtue of their parenthood, could meet their death before the age of 20, there would be a marked anticipation of the age of death in the offspring. This antedating, however, of the age of accidental death in the children, which is comparable to the antedating of the age of onset of mental disease, can have nothing to do with any law of inheritance, but only with the fact of parenthood.

This objection relating to parenthood applies particularly to the sphere of mental disease, for if we consider the commonest form of mental disease, schizophrenia, we find that according to Kraepelin two-thirds of the cases occur before the age of 30. It is therefore only to be expected that this common form of mental disease, whose onset is early, will be much less common in those who are already parents.

With regard to Mott's conclusion that the disease occurs in a more intense form in the offspring, the explanation again depends on the fact of parenthood. Since the members of the older generation must be parents, they are unlikely ipso facto to suffer from the severe kinds of mental disease which occur in the first three decades of life, such as congenital imbecility, and the more malignant forms of dementia præcox. If they do suffer from dementia præcox it will not be so rapidly progressive or lead to such a profound dementia. It is clear then that to say that the disease occurs in a more intense form is merely to say that it occurs at an earlier age.

If reference is made again to fig. 1, it will be seen that the percentage of cases of mental disease which occur in the first two decades of life is apparently much higher among children of the insane than among the

general run of those admitted to mental hospitals. This phenomenon cannot be explained by the fallacy of parenthood described above, as Mott urged in defending himself against Pearson's criticism. A brief consideration, however, of the method in which the material was collected will show how there was a selection of material which gave chiefly those children who became affected at an early age.

In the first place, an enquiry regarding relatives belonging to different generations who are both admitted to mental hospitals will lead to a selection of those cases which are admitted about the same time or within a decade of each other. As parents are on the average 30 years older than their children, those who are admitted to hospital in the same year as their children will show an anticipation averaging 30 years. Those cases, on the other hand, in which the child becomes insane at an age 30 years older than that at which the parent became insane will almost certainly not be included, because the child is admitted to hospital on an average 60 years later than the parent, and so will not be noticed.

Mott himself, in trying to meet another objection to be described later, stated that the bulk of his cases of parent and child were admitted within 15 years of each other, and I find that the average interval between admissions for the Colney Hatch material is 13·7 years.

If we now consider three hypothetical cases, we can see how Mott's selection of material favours his theory (Table 1). In the first example,

TABLE 1.—HYPOTHETICAL FAMILIES SHOWING HOW THE SHORT TIME BETWEEN ADMISSIONS OF PARENT AND CHILD LEADS TO A SELECTION OF CASES SHOWING GREAT ANTICIPATION.

	A	B	C
Parent			
Born	1830	1830	1830
Year of onset	1890	1890	1860
Age at onset	60	60	30
Child			
Born	1860	1860	1860
Year of onset	1890	1920	1920
Age at onset	30	60	60
No. of years between two admissions ...	0	30	60
Amount of anticipation	30	0	−30

parent and child are admitted in the same year and there is anticipation of 30 years; in the second the child is admitted 30 years after the parent, and there is no anticipation; in the third the child is admitted 60 years after the parent, and there is postdating of 30 years. If all three cases were recorded, there would on an average be no anticipation, but since on an average Mott

only records those cases admitted within 14 years of each other, the probability is that only the first case will be recorded, namely, that which shows marked anticipation. The accompanying Table (Table 2) shows how many of the children were admitted either before or in the same year as their parents, and how many within 10, 20, and 30 years. The children who were admitted 30 years after their parents were on the average admitted at an age greater than that of their parents when admitted, and so there is on the average postdating. There is no need to suppose that the scarcity of those admitted 30 years after their parents depends on anything else than the method of collecting the material, for, as Mott himself states, the bulk of the cases were admitted within 15 years of each other.

TABLE 2.—SHOWING RELATION OF INTERVAL BETWEEN ADMISSIONS OF PARENT AND CHILD TO AMOUNT OF ANTICIPATION.

No. of years that child was admitted after parent	No. of cases	No. of cases of postdating	Per cent. of cases of post-dating	Anticipation in years
Less than 1	108	0	0	35·1
1-10	113	0	0	23·3
11-20	102	1	1	13·3
21-30	89	13	15	4·9
31-40	40	23	58	−1·1
Over 40	30	30	100	−16·6

As we have seen, Mott has given preference for inclusiòn in his material to those children who became insane about the same time as their parents. Now, in the matter of age, new patients whose parents have recently been admitted to a mental hospital cannot be said to constitute a fair sample of the general admissions. They will be much younger, just as those who are parents will be much older, than the general run of admissions.

In thus selecting his material Mott was neglecting all those members of the second generation who, though sane at the time of the investigation, became insane later; yet it was just those cases, as we have seen, which became insane long after their parents, that told against the theory of anticipation. Now, some of the insane children of insane parents recorded by Mott in 1911 had younger brothers and sisters who were sane in 1911, but who have since become insane. If we include these children, the amount of apparent anticipation will be much less. I have found 20 families in the records at Colney Hatch in which psychotic parents had had one or more psychotic children by 1911, these being included in Mott's material, and in

which one or more children have since become affected. Taking only the children recorded by Mott, there is anticipation of 25 years, but taking only the younger members of these families, who have been admitted since 1911, there is an anticipation of only 13 years (Table 3). These figures, then, show how this fallacy alone contributes largely to the apparent anticipation.

TABLE 3.—SHOWING FAMILIES IN WHICH SOME SIBLINGS WERE AFFECTED BEFORE 1911 AND SOME SINCE 1911. THE AMOUNT OF ANTICIPATION HAS DECREASED WHEN THE LATTER ARE TAKEN INTO ACCOUNT.

Number of parents	20
Number of offspring	53
Anticipation in 1911	25 years
Anticipation in offspring alone who have become psychotic since 1911	13 years

It follows, therefore, that if we are to compare the age at which members of an older generation become insane with that at which members of a younger generation become insane, we must wait till all the members of the latter are past the danger zone for insanity. Only in this way shall we be certain that we are not counting as sane those who will later become insane. If we take as our younger generation those who were born on an average in 1860, we shall largely avoid this pitfall, for to say that a man of 72 is free from all possibility of insanity has quite a different significance from saying that a man of 32 is free. The importance of this point was apparently not fully appreciated by Mott, for the average age of his younger generation was 26.

This particular fallacy can very easily deceive one when examining a pedigree into thinking that evidence of anticipation is presented in it. In the *Archives of Neurology and Psychiatry* (London), vol. VI, two of Mott's pupils, H. W. White[11] and J. C. Wooton[12], together published 35 pedigrees in which anticipation was for the most part apparently demonstrated. In nearly every case the investigator took as his starting point (propositus) an insane patient of about 25, in the preceding generation of whose family was a fair sprinkling of mental disease. Now if we include, as Mott did, the acquired forms of mental disease, such as general paralysis and alcoholic psychoses, four times as many cases of mental disease occur after the age of 25 as before it. About four out of every five members of the preceding generation will therefore have become insane after that age. In the younger generation, however, the average age is about 25, and so this is impossible. If insanity occurs in the younger generation, this is said to be a case of marked anticipation, and if it does not, this is said to be a case where the stock is mending. The real truth, however, is that, if one waited till the patient's generation had all become extinct, one would no doubt find that the same number had become insane in later life as in the previous generations.

This is shown clearly by reference to these very pedigrees. In Table 4, I have taken as the younger generation that which preceded the propositus, and compared it where possible with the preceding generations. It will be seen that there are five cases of antedating and four of postdating, and that the balance in years is on the side of the latter.

TABLE 4.—SHOWING AVERAGE AGES AT WHICH MEMBERS OF PARENTS', GRANDPARENTS' AND GREAT GRANDPARENTS' GENERATION BECAME PSYCHOTIC IN PEDIGREES CITED IN SUPPORT OF ANTICIPATION BY H. W. WHITE AND J. C. WOOTON.

(Numbers in brackets refer to number of persons psychotic in each generation.)

		Great grand-parents' generation	Grand-parents' generation	Parents' generation	Average antici-pation
White	Pedigree No. 4	50 (1)	40 (1)	52 (1)	−2
	,, ,, 6	42 (1)	50 (5)	25 (7)	17
	,, ,, 10	0 (1)	68 (2)		−68
	,, ,, 11		55 (1)	54 (1)	1
	,, ,, 16	60 (1)	72 (1)		−12
Wooton	,, ,, 2		44 (1)	38 (4)	6
	,, ,, 3		28 (1)	35 (5)	−7
	,, ,, 4		25 (2)	20 (3)	5
	,, ,, 7		20 (1)	11 (2)	9

There are thus—
5 cases of anticipation, average 7·6 years.
4 ,, ,, postdating, ,, 20·2 years.

One of the authors, H. W. White, remarked at the conclusion of his paper that the amount of anticipation was smaller in the earlier generations. He did not, however, realize that the apparent anticipation in the last generation was on this explanation spurious.

CASES OF INDIRECT HEREDITY.

In addition to examining the amount of the difference in age of onset as between parent and child, Mott examined the cases of all those related as nephew or niece to uncle or aunt. Here he found anticipation, but to a lesser degree (in the Colney Hatch material it is 13·4 as against 16·0 years); and this he attributed to the fact that the relationship was less close. That there was some anticipation, however, he attributed again to the fact that those related collaterally belonged to a stock in which there had set in a degenerative process, in virtue of which the members of each succeeding

generation became affected at an earlier age, until propagation was no longer possible.

A much simpler explanation, however, presents itself. The fallacy created by comparing those who must be parents with those who need not be parents holds only partially for uncles and aunts. The apparent anticipation is therefore less. Incidentally, examination of the material in this piece of work shows a regrettable oversight, for included among the uncles and aunts were those who were no blood relations, but related only by marriage. At first sight it might be thought that to include persons who were no relation would decrease the amount of anticipation, but the contrary holds. In order to be an uncle or aunt by marriage, it is necessary to be at least in the neighbourhood of 20 years of age, and so the same exclusion of members of the older generation from the early forms of mental disease obtains in this case too.

Table 5 shows how the selection of material has influenced Mott's results in relation to uncles and aunts. The Table shows the amount of anticipation

TABLE 5.—SHOWING RELATION OF INTERVAL BETWEEN ADMISSIONS OF UNCLES OR AUNT AND NEPHEWS OR NIECES TO AMOUNT OF ANTICIPATION.

No. of years that nephew or niece was admitted after uncle or aunt	No. of cases	No. of cases of postdating	Per cent. of cases of post-dating	Anticipation in years
Less than 1	38	6	16	17·9
1-10	66	0	0	19·1
11-20	63	9	14	11·8
21-30	46	12	24	5·3
31-40	32	20	60	0
Over 40	22	21	96	−14·1

in relation to the length of time between admissions. It will be noticed that in contrast to the parent-and-child Table, very few nephews or nieces apparently became insane before their uncles or aunts. Thus only 38 were admitted previously or in the same year, as against 66 who were admitted in the decade following the admission of uncle or aunt. The obvious reason for this is that relatives are much less likely to record that a patient has an insane nephew than that he has an insane uncle. Where, therefore, the uncle is admitted first, the case is not likely to be noticed. This omission of cases where very young nephews and nieces became insane partly accounts for the fact that apparent anticipation between uncles or aunts and nephews or nieces is not as great as between parents and children.

OTHER FACTORS FAVOURING SPURIOUS ANTICIPATION.

There are other fallacies which help to increase the amount of apparent anticipation. For instance, Mott counted, as the year of onset of the disease, the year in which the patient was first admitted to an L.C.C. mental hospital. Now in the parents' generation patients were for the most part only admitted when they were dangerous to the community or quite helpless. As Mott himself pointed out, between 1890 and 1910 the accommodation for the insane in the L.C.C. asylums was almost doubled, in order to contain the large proportion of insane who were either roaming at large, or else, being helpless, were nursed in ordinary hospitals.

In going over the records of the older generation, I repeatedly found notes stating that the patient had been previously insane, but as he had not been admitted to an L.C.C. asylum, the later attack was counted as being the first.

This greater accommodation of the insane in L.C.C. asylums (which amounted to 27,000 in 1910 compared with 16,000 in 1890) made possible the admission of patients at an earlier age. Not only was there more room for the mentally diseased in asylums, but the asylums became more like hospitals. Relatives have therefore shown less reluctance in sending the patients in for treatment, and so they have come to be admitted at an earlier age.

It was to meet this objection that Mott stated that the parents and children in his material were admitted for the most part within 15 years of each other. He appears, however, to have overlooked the fact that such a selection of material could only lead to an apparent antedating in the children which was spurious. Parents are on the average 30 years older than their children. If the children included are admitted on an average 14 years after their parents, they must be 16 years younger at the time of their admission. As we have seen, children who are admitted at a later age than their parents, are admitted, on an average, more than 30 years after their parents. These, however, were extremely unlikely to be included in Mott's material.

Even if all the objections to Mott's theory which have so far been put forward were invalid, it could still be shown from analysis of his own material that he failed to prove any law of anticipation such as he described. If his theory were correct, we should find that the amount of anticipation would have no relation to the age of the parent, but would remain the same whatever the parent's age. Thus, if the average amount of anticipation were 20 years, parents admitted first at 70 would, on the average, have a child who became affected at 50, the parents who became psychotic at 50 would have on the average a child who became insane at 30, and if the parents first admitted at 30 had children, these would, on his theory, be mostly feebleminded. If, however, the material has been so selected as to include chiefly cases admitted about the same time and where, therefore, the onset of insanity

was late in the parents and early in the children, we shall find a different result. The amount of anticipation will not always be the same, whether the parent became insane at 70 or 60 or 50 or 40. We shall find on the other hand, that as the age at which the parent became insane decreases, so also will the amount of anticipation decrease, so that the children of parents who became insane in early life will show not antedating but postdating. This is in fact what we do find.

TABLE 6A.—AMOUNT OF ANTICIPATION IN RELATION TO PARENTS' AGE.

Age of parent	No. of cases	Average age of parent	Average age of child	Anticipation in years	No. of cases of postdating	Per cent. of cases of postdating
Over 70	41	74·6	40·6	34·0	0	0
60-69	101	63·6	37·2	26·4	2	2
50-59	105	53·8	31·5	22·3	5	19
40-49	99	44·9	33·4	10·5	21	21
30-39	95	34·2	29·1	5·1	27	27
20-29	33	27·9	30·6	−2·7	12	36
0-19	8	16·0	26·8	−10·8	5	63

If reference is made to Table 6A it is seen that the amount of anticipation decreases from 34·0 years for parents admitted over 70, to 5·1 years for parents admitted between 30 and 40, and where parents become insane below the age of 30, it is actually more usual for their children to show postdating, not antedating.

TABLE 6B.—AMOUNT OF ANTICIPATION IN RELATION TO AGE OF CHILDREN.

Age of children on admission	No. of cases	Average age	Average age of parent on admission	No. of cases of postdating	Per cent. of cases of postdating	Average anticipation
Under 20	65	16·8	47·4	3	5	30·6
20-30	143	24·3	50·0	7	5	25·7
30-40	115	34·3	50·6	43	37	16·3
40-50	81	44·2	53·0	23	28	8·8
50-60	39	53·6	55·5	23	60	1·9
60-70	14	64·6	55·7	7	50	−10·9
Over 70	5	70·4	50·0	5	100	−20·4

Similarly, Table 6B shows that where we do find children who became insane as late as 50, 60 per cent. of their parents became insane at an earlier age, i.e., postdating is again the rule.

Tables 6c and 6d show similar findings for indirect heredity.

TABLE 6c.—Amount of anticipation in relation to uncles' and aunts' age.

Age of uncle or aunt	No. of cases	Average age of uncle or aunt	Average age of child	Anticipation in years	No. of cases of post-dating	Per cent. of cases of post-dating
Over 70	6	76·5	24·2	52·3	0	0
60-69	27	62·1	32·9	29·2	1	4
50-59	47	53·2	33·1	20·1	3	6
40-49	50	43·4	30·3	13·1	8	16
30-39	72	34·0	28·0	6·0	20	28
20-29	50	24·5	26·7	−2·2	23	46
0-19	18	17·4	23·2	−5·8	13	72

TABLE 6d.—Amount of anticipation in relation to age of nephews and nieces.

Age of nephews or nieces	No. of cases	Average age for 10-year period	Average age of uncle or aunt	No. of cases of post-dating	Per cent. of cases of post-dating	Average anticipation
Under 20	48	16	33·5	1	2	17·5
20-30	99	24·4	38·7	14	14	14·3
30-40	71	33·6	44·4	20	30	10·8
40-50	28	43·5	39·7	18	64	−3·8
50-60	15	54·1	44·4	10	67	−10·0
60-70	0					
Over 70	2	70·5	62·0	2	100	−8·5

Figure 2 shows the age-incidence of children on admission when the parents were aged over 70 on admission, aged 60 to 70, 50 to 60 and so on.

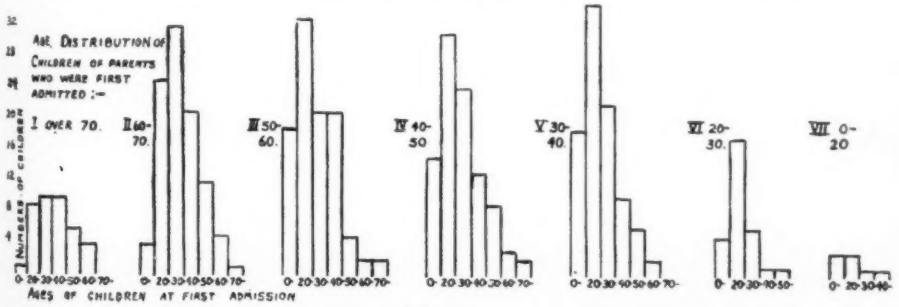

Fig. 2.

It will be noticed that the mean age of children on admission when the parents were aged 60 to 70 was 30 to 40, that is, 30 years younger. Similarly for parents of 50 to 60, there is a mean anticipation of 30 years. This, however, is the result of the selection of cases of parent and child admitted about the same year, for in such cases the parent will be 30 years older than the child. Similarly, in the case of uncles and aunts, those who were over 60, over 50 and over 40 on admission show a mean anticipation of 20 years. This again is caused by the selection of material, but the amount of the anticipation is less, because many uncles and aunts become mentally diseased before the age of 20, whereas parents as we have seen can scarcely do so.

FRATERNAL ANTICIPATION.

With regard to socalled fraternal anticipation, although Mott collected material, he did not, so far as I am aware, publish any results. In the present material I examined the records of 425 pairs of siblings, and found that the older sibling became insane at an age 4·1 years greater than the younger sibling. By way of control, however, I examined 41 pairs of cousins and second cousins and found that the older cousin became insane at an age 4·0 years older than the younger cousin. The difference is therefore almost identical in the control series.

The apparent anticipation again results from the way in which the material is collected. Let us take a case where one sibling is four years older than the other. If both are admitted in the same year, then this case which shows fraternal anticipation of four years is certain to be noticed. If, however, there is a case where the younger is 4 years older at the time of admission than the older sibling was at the time of his admission, then there will be a lapse of eight years between the two admissions and this case is not so likely to be noticed. There is therefore a selection of material which favours apparent anticipation.

Another factor which favours apparent anticipation is that relatives put off as long as possible the admission of the older sibling who becomes insane, but if an older brother is already in a mental hospital, the second is likely to be admitted when the first symptoms appear. Thus where two siblings really both become affected at 20, the probability is that the older will be admitted when he is 21 or 22, whereas the younger will be admitted at 20.

From the above material, an attempt was made to separate the cases of schizophrenia from those of the manic-depressive group. It was difficult to be sure of diagnosis, as this was in many cases made soon after admission, and schizophrenics in the earlier records might be labelled mania, stupor, delusional or confusional insanity, or melancholia, while manic-depressives were often diagnosed as cases of stupor, confusion, or delusional insanity. Taking, however, the cases where the diagnosis was pretty clear, the following figures were obtained.

Of 153 pairs of manic-depressive siblings, 102 showed average fraternal antedating of 10·4 years, while 51 showed average postdating of 7·5 years. The average antedating for the 153 was 4·4 years.

Of 36 pairs of schizophrenic siblings, 24 cases showed average antedating of 6·0 years, while 12 cases showed postdating of 5·8 years. The average antedating for the 36 was 2·1 years.

Little importance, however, can be attached to these figures, since in the control series there was an anticipation of 4·0 years.

If we consider those families where three, four, or five siblings were insane, we find that there is a fairly marked apparent anticipation between the first and second but much less between any other two. This probably results from the fact mentioned above that after one sibling is admitted there is no hesitation on the part of relatives about admitting others. Thus in 35 cases where three or more siblings became insane, there was an average anticipation of 2·7 years of the second over the first, but only of 1·4 years of the third over the second.

If we examine the few families where four or five siblings become affected we find much less evidence of anticipation because the chief statistical fallacies described do not hold. We are not counting as sane those who will later become insane.

The following Table shows four cases of families where four siblings became insane and two where five siblings became insane.

The roman numerals indicate order of birth, the arabic numerals the age at onset. The diagnosis in each case is also given. At the bottom are given the average ages at onset.

TABLE 7.—CASES OF FAMILIES IN WHICH SIBLINGS BECAME INSANE.

Order of birth	I	II	III	IV	V
Age at onset and diagnosis	39 melancholia	36 schizophrenia	28 melancholia	24 melancholia	37 schizophrenia
	15 mania	13 mania	15 mania	23 mania	16 mania
	31 melancholia	0 imbecile	34 paranoia	23 mania	
	0 imbecile	23 mania	38 melancholia	22 paranoia	
	29 schizophrenia	20 melancholia	21 schizophrenia	20 melancholia	
	15 melancholia	23 mania	17 schizophrenia	29 schizophrenia	
Total age	129	115	153	141	53
Average age at onset	21·6	19·2	25·5	23·5	26·5

It should be mentioned here that Rüdin[19] examined 72 cases of schizophrenic siblings and found that the older sibling was affected on an average at the age of 25·86 years while the younger's age at onset was 24·68.

There was therefore a difference of only 1·18 years between the ages at onset and Rüdin himself considers the anticipation to be spurious for the reasons mentioned above. Two English text-books, however, quote Rüdin as believing these figures to support the theory of anticipation.

CRITICISM OF MOTT'S CONCLUSIONS.

It now falls to examine the conclusions which Mott drew from his statistical data. He believed that the apparent earlier date of onset of mental disease in the younger generation was of great importance, and that the disease was more intense in form. He believed that this was one of nature's methods for ending or mending a degenerate stock. We have seen, however, that there is no evidence that nature eliminates a stock by the gruesome method of causing the disease to appear at an earlier age in each succeeding generation. Nor has Mott brought forward evidence that insanity and sterility go hand in hand and that therefore it is superfluous for the eugenist to dissuade the insane from marriage. The literature contains numerous pedigrees which show that tainted stocks can be exceedingly prolific.

Mott also believed that his figures ' were of great importance to the physician, because he can say there is a diminishing risk of the child becoming insane after the age of 25, a matter of great importance in the question of marriage.'

We have seen, however, that there is nothing in Mott's material which would lead us to suppose that the children of insane parents differ from the general run of mental patients in regard to age at onset. Now, in the latter case, if we include the types of psychosis which are caused almost entirely by exogenous factors, such as general paralysis and alcoholism, we find that for every one person who becomes insane before 25, about four become insane after that age. These figures of 4 to 1 were quoted by Dr. D. Heron in his criticism of Mott. If Dr. Heron were a psychiatrist and not a statistician, he would have excluded the acquired psychoses, and said that as far as heredity goes, the chance of becoming insane after 25, is only about twice what it is before that age. His figures 4 to 1, however, were justified as an argumentum ad hominem, for Mott, when he wrote, included general paralysis in his material.

The question of the age at which mental disease comes on in members of a tainted family no longer influences the psychiatrist in giving advice about marriage so much as the question of what type of psychosis is found in that family. During recent years valuable work has been done to show the probability of mental disease occurring in the direct and indirect descendents of psychotics in comparison with those of healthy individuals.

We can say for instance that whereas an individual whose parents are healthy has only a 0·68 per cent. chance of becoming schizophrenic, the child of a schizophrenic parent has an 8·4 per cent. chance. Similarly for manic-depressives, the figures are 0·38 per cent. for those with normal parents but 31·4 per cent. for those with manic-depressive parents. The psychiatrist can merely state what the prognosis is and leave it to the patient to make the decision. The figures (Table 8) quoted are taken from Hoffmann[3] and they are being substantiated by further work in Germany and elsewhere.

TABLE 8.—SHOWING PROBABILITY OF MENTAL DISEASE IN CHILDREN, ONE OF WHOSE PARENTS SUFFERS FROM ITS THREE CHIEF FORMS, COMPARED WITH AVERAGE POPULATION (HOFFMANN, 1928).

	Psychotic child of psychotic parent Per cent. affected	Average population Per cent. affected	Greater probability of these diseases in the former
Schizophrenia	8·4	0·68	12·3 times
Manic-depressive psychosis	31·4	0·38	82·6 times
Epilepsy	9	0·29	3·1 times

Lastly, Mott says that his figures are of importance in connexion with the subject of social surgery of the insane, for when the first attack of insanity occurs in the parent, the children for the most part have all been born. In this connexion, it may be pointed out that the form of mental disease which is most strongly inherited is the manic-depressive psychosis, which in virtue of its recurrent nature allows the patient to become a parent during the intermissions. There is nothing in Mott's results which should prevent a psychiatrist from discouraging a patient suffering from, say, attacks of recurrent mania from becoming a parent.

In conclusion, it would seem then that the onset of mental disease in each succeeding generation of a tainted stock at an earlier age and in a more intense form, so far from being the rule, is exceeding difficult to demonstrate in any pedigree, and that where it does occur, it probably depends on a combination of Mendelian factors, the exact nature of which is still a matter for investigation. The results obtained by Mott do not make it necessary to postulate any law in virtue of which nature ends or mends a degenerate stock. The facts are capable of being explained more simply in the way described above. It rests with those who believe in the law to adopt a different method of approach to prove their case.

SUMMARY.

Many writers have remarked that in cases of hereditary disease the disease appears to occur at an earlier age in each succeeding generation. This has been supposed to be the expression of a hereditary law, in virtue of which a tainted stock comes to an end. This law was described by Morel as occurring in mental disease. Sir Frederick Mott sought to prove its existence by examination of material in the London County Mental Hospitals. In the present study, a great deal of this material has been brought up to date and examined. Arguments are brought forward to show that the apparent antedating of mental disease in the children is merely the result of the way in which the material was collected. Similar arguments explain the apparent antedating of mental disease in younger siblings compared to older ones. Conclusions based on the supposed law of anticipation have been applied by Mott and others to the sphere of eugenics. These conclusions are criticized.

I should like to express my thanks to Dr. F. L. Golla, Director of Research for the London County Mental Hospitals, for allowing me to undertake this study; to Dr. J. Brander, Superintendent of Colney Hatch, for granting me access to this material; to Dr. Aubrey Lewis for valuable advice and information; and to Dr. Bruno Schulz, Munich, for references to German work on the subject.

Part of this work was done during the tenure of a Pinsent-Darwin Research Studentship from Cambridge University.

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CEREBROMACULAR DEGENERATION

WITH CLINICAL AND PATHOLOGICAL NOTES OF A CASE.*

By

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AFTER amaurotic family idiocy had been first recognized for its ocular changes in 1881 by Warren Tay, Batten, in 1903, published a description of a disease which he described as 'cerebral degeneration with symmetrical degeneration in the maculæ.' This condition he regarded as having some of the characteristics of Tay-Sachs disease but differed from the latter in that: (1) it was not limited to Jews; (2) there were not the same fundus findings; (3) the first signs began at a later age; and (4) the progress was slower. Since these original descriptions the apparent gap between the two diseases has been bridged by the recording of cases (known as the late infantile form of amaurotic family idiocy) occurring between the age-periods of Tay-Sachs disease and of cerebromacular degeneration. The bridge has been strengthened by the fact that findings which had been described as typical of one form of the disease were reported as occasionally appearing in the other. Holmes and Greenfield state that the pathological change is 'a special form of degeneration of the cell-body associated with the deposition in it of a peculiar form of lipoid.' Often the axon undergoes secondary degeneration, but it may remain intact even when its cell-body is damaged.

Sachs describes the cell-degeneration as tending to pass through the following stages: (1) huge increase in size, (2) vacuolization, (3) disappearance of the nucleus, (4) distortion of the entire cell-mass, (5) swelling of dendrites. He regards this degeneration as tending to affect every ganglion-cell in the nervous system.

The precise nature of the disease-process is still in dispute. Sachs, Bielschowsky and others regarded the condition as a hereditary one associated with primary changes in mesodermal tissues. They say that there is a surprising relationship with (1) Gaucher's disease and with (2) the familial infantile disease occurring in Jews and closely resembling Gaucher's disease, called lipoid cellular splenohepatomegaly or 'Niemann-Pick disease.' Both are said to be variants of general lipoid degeneration, with which they group

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Tay-Sachs disease and cerebromacular degeneration. Mixed cases are said to have been observed. Lipoids from degenerating mesodermal tissue, predominantly the reticulo-endothelial system, are, on this hypothesis, presumably deposited in the ganglion-cells.

Schaffer denies this relationship and regards Tay-Sachs disease and cerebromacular degeneration as being a germ-layer disease with a primary ectodermic selectivity, i.e., a primary degeneration of the ganglion-cells of the nervous system. He finds microscopical differences in the nervous system between Tay-Sachs disease and Niemann-Pick's disease.

CLINICAL CASE.

D. I., male, aged six years at time of death. He was aged four years and four months when first admitted to hospital, on June 22, 1928. The patient was a full-term child of non-Jewish parents. There was nothing of note in family history, an elder brother being alive and well. He sat up at five months of age, walked at 17 months, and talked at 18 months. Except for being somewhat retarded in development and the gait being clumsy, the patient showed no obvious signs of illness until the age of three years, when in March, 1927, he had a convulsive seizure, with loss of consciousness for about a quarter of an hour. Similar attacks followed at about fortnightly intervals, during which it was noted that the left arm was drawn up. At three and a half years of age his gait was so clumsy that he could not walk without support. Vision was progressively impaired.

On examination (May, 1928).—The only physical sign observed was a slight degree of spasticity of the left arm and leg, and it was noticed that there was a wandering look about the eyes.

On admission (June 22, 1928).—Mentality impaired. Although up to the end of 1927 he had been able to discern pictures, he now appeared to be quite blind. The pupils were equal and reacted to light. A primary optic atrophy was present with diminished size of the vessels, and depigmentation and 'peppering' of the retina as in complete degeneration, but there was no cherry-red appearance of the maculæ. Hearing appeared normal, and the boy always smiled when a shrill whistle was blown. At first he seemed to recognise his own name, but later even loud sounds produced no reaction. Cold air blown into the right auditory meatus produced nystagmus in 30 seconds, and into the left ear in 40 seconds. Other cranial nerves were normal. Cutaneous sensation, so far as it could be explored, appeared to be normal. The patient did not speak, but occasionally made a noise.

Jerky movements were frequent, chiefly on the left side, especially of the left hand and arm. All tendon-reflexes were present and equal on the two sides. The plantar responses were at first somewhat doubtful in character, but a month later were definitely extensor.

The cerebrospinal fluid was clear, not under pressure, showed less than 1 small lymphocyte per c.mm.; total protein 0·02 per cent.; no excess of globulin; and a Lange's colloidal gold curve 0111000000, with a negative Wassermann reaction both in the fluid and in the blood.

During his stay in hospital, the patient developed measles, from which he recovered.

Three months after admission, convulsive attacks of generalized clonic spasm became more frequent, with loss of consciousness for a few seconds, the eyes

open and fixed, the arms flexed, and the legs extended and rigid, without any biting of the tongue or incontinence.

The condition continued more or less as already described throughout 1928 and 1929, and then gradually became worse, with continuous generalized myoclonic contractions. Towards the end of 1929, the attitude of complete flexion, with increasing rigidity, was adopted. The patient was discharged from hospital on December 23, 1929, but died at home of bronchopneumonia eight days later.

A post-mortem examination was carried out by Dr. Stanley G. James, to whom we are indebted for the following notes.

PATHOLOGY.

Post-mortem Examination.—The body was moderately emaciated, with considerable post-mortem discoloration of the skin, and with extensive bed-sores over the buttocks and sacrum.

Head.—The skull-cap was thick and heavy for a child of this age; anterior fontanelle closed; shape asymmetrical with bulging of left parieto-occipital and right frontal regions; dura moderately adherent to bone. The superior longitudinal sinus contained a firm dark brownish-red thrombus, extending to the torcular but not into the lateral sinuses—all the surface veins on the superior convexity of the brain being distended and firm, and giving a dark purplish appearance to the brain. The base of the skull showed no obvious adnormality.

The *spinal cord* and several of the larger nerve-trunks to the limbs were removed for examination, nothing of importance being noted in their naked-eye appearances.

No remains of the thymus were found.

The *heart* showed great dilatation of right auricle and ventricle, which were distended with agony thrombus, but there was no endocarditis. The bronchi contained much mucopurulent exudate, and the lung-tissue was congested, with some patchy bronchopneumonia at the bases, but no generalized pneumonic consolidation. Beyond some congestion and cloudy swelling combined with some fatty degeneration of the liver and kidneys, nothing of note was found in the abdominal organs, the *spleen* being recorded as showing no obvious abnormality. The immediate cause of death appeared to be the cerebral thrombosis and bronchopneumonia following upon septic absorption from bed-sores.

Further examination of the brain after fixation in 10 per cent. formol-saline.—In addition to the thrombosis of the great longitudinal sinus and its main tributaries from the surface of the hemispheres, there was also recent red thrombus in the left internal carotid and middle cerebral arteries. The whole brain was distinctly small with general diminution in the size of the convolutions, particularly those of the occipital lobes, with corresponding widening and exaggeration of the intervening sulci. The diminished size of the cerebellum was particularly noticeable, giving it a shrivelled or

' miniature ' appearance, its individual folia being small and narrow (fig. 1). On horizontal section through the hemispheres, the lateral ventricles were considerably dilated, evidently owing to retraction of the atrophic brain-substance, the white matter being considerably reduced in amount, and the grey matter of the atrophic cerebral cortex being distinctly narrowed. The third and fourth ventricles and the aqueduct of Sylvius were also considerably dilated, and the small size of the cerebellum appeared to be due mainly to atrophy of cortical grey matter in rather greater degree than to atrophy of the white, though this also was much reduced in volume. The white matter of the cerebral hemispheres was distinctly greyer than normal, contrasting less markedly with the grey matter, but the change was more or less uniform and without the patchy character seen in Schilder's periaxial encephalitis, the characteristic sparing of the subcortical arcuate fibres typical of the latter disease not being here in evidence.

FIG. 1.—Naked-eye view of cerebellum (natural size) to show atrophy, especially of the cortical folia.

Histological Examination: Peripheral Nerves.—From the left arm, the median, and from the left leg the sciatic and internal and external popliteal nerves were examined, both in longitudinal and transverse section. All showed a moderate degree of fibrosis, mostly around the main funiculi, with patchy old-standing degeneration of the fibrils, advanced in some parts of the nerves, especially in the external popliteal, and, to a lesser degree, in the median. There was no recent fatty degeneration of the myelin sheaths with Marchi, but there was a considerable increase in the numbers of the neurilemma nuclei.

Spinal Cord.—The white matter throughout did not show any very obvious changes—no appreciable degree of recent fatty degeneration of myelin or older demyelination. The direct pyramidal tracts, especially in the dorsal region, appeared to be rather smaller than normal; whilst the posterior columns throughout were perhaps a little larger in proportion to the slightly reduced remainder of the cord. All the nerve-cells, but especially those of

the anterior horns, showed varying degrees of degeneration throughout the whole cord, but most markedly in the lumbar region. The margins of many of the cells were indistinct, and the cytoplasm of many showed a clear hyaline swelling. In paraffin sections stained by van Gieson's method this globose swelling stained a faint diffuse yellowish colour, while some finely granular protoplasm usually survived around the peripherally placed nucleus. In

FIG. 2.—General low power view of the rolandic cerebral cortex showing spongy appearance of the middle and deeper zones of the cortex in paraffin section. Compare figs. 3 and 4, the former a high-power view of the area in this section marked with the bracket; the latter a frozen section in which the nerve-cells are not so contracted and vacuolated. (Hæmatoxylin-eosin).

the dorsal region and in the lumbar enlargement these changes showed varying degrees of advancement up to complete disappearance of the nucleus, with only a clear, more or less shrivelled and yellowish necrotic-looking remnant of the cells surviving, and in practically all the cells the Nissl-granules had completely disappeared.

Cerebrum : Cortex.—The surface vessels were engorged, but there was no cellular infiltration around them or in the leptomeninges. In paraffin sections the grey matter of the middle and deeper zones had a general spongy appearance (fig. 2). The ganglion-cells looked small, rounded or irregular, and shrunken, with the nucleus retracted to one side but usually staining fairly well and surrounded by a zone of granular cytoplasm of varying extent. The latter was generally scanty and in some cells almost absent, the remainder of the cell-body appearing at first sight like a clear space or vacuole to one side of which lay the nucleus and the surrounding residual

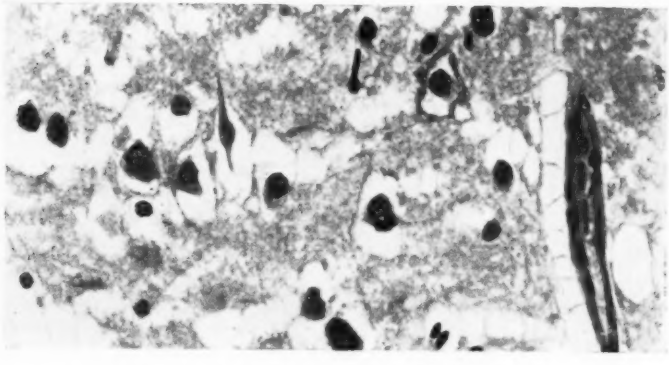


FIG. 3.—High-power view of area marked with bracket in fig. 2, showing the small retracted appearance of the nerve-cells in paraffin sections, the nuclei, surrounded by a little granular protoplasm, being retracted to one side of vacuole-like spaces. (Compare with frozen section of a similar area shown in fig. 4). (Hæmatoxylin-eosin.)

protoplasm. This clear vacuole-like space, however, had usually a fairly definite border, and in frozen sections was found to be filled with somewhat granular to more or less homogeneous-looking greyish material suggestive of coagulated fluid. The granules around the nucleus stained pinkish-orange with Sudan III. For convenience of description only, the general appearance might be likened to that of a coarsely granular eosinophil myelocyte stained with Leishman (figs. 3 and 4). In the grey matter glia-cells were not increased but rather diminished in numbers, while comparatively few ' parasitic ' small round cells could be seen around the ganglion-cells. The spongy appearance of much of the cortex was due to tangential sections of the swollen cytoplasm of the nerve-cells. No cellular infiltration around capillaries was seen.

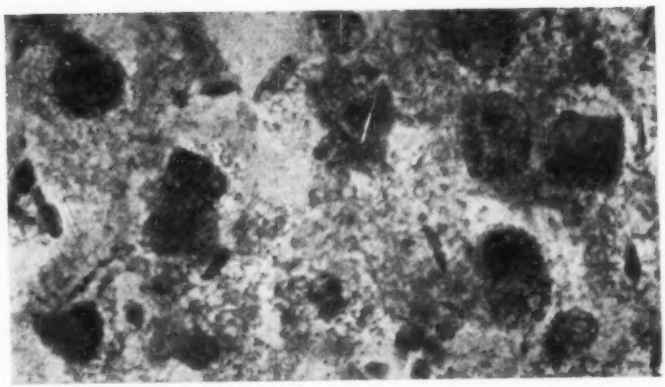


FIG. 4.—High-power view of a similar area to that shown in fig. 3. The magnification is the same, but the section is a frozen one and the nerve-cells, etc., do not show the retraction seen in the paraffin section. The granules in the cytoplasm are stained pinkish-orange with Sudan III, and the vacuole-like spaces are less exaggerated and contain more or less amorphous material, perhaps fluid or semi-fluid coagulated by the formalin fixation. (Sudan III–hæmatoxylin.)

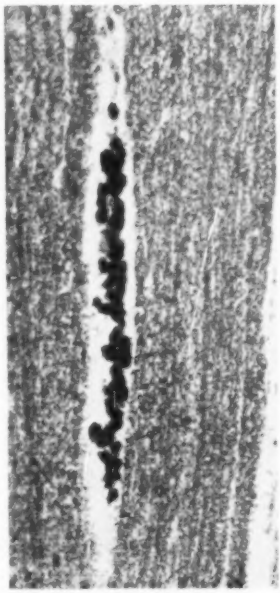


FIG. 5.—White matter of cerebral hemisphere. Compound granular corpuscles containing fat, stained with Sudan III, lying in perivascular space. Frozen section.

White Matter.—In contrast to the grey matter of the cerebral cortex, in the white matter of the cerebrum were seen numerous small glia-cells and also small lymphocyte-like cells diffusely distributed. Capillaries and small venules stood out clearly with well stained endothelial walls. These appeared unduly numerous and were mostly distended with red blood corpuscles. Dilatation of the perivascular spaces was not a very marked feature, but occurred in moderate degree, with also a varying amount of perivascular cellular infiltration, mostly minimal or absent. Only slight fatty degeneration of the capillary endothelium was demonstrated by the Marchi method, but with Sudan III in frozen sections occasional clumps and masses of fat-containing compound granular corpuscles were found in the perivascular spaces, especially at the junctions of capillaries with small veins, where also the lining endothelium sometimes contained localized collections of fat globules and granules in considerable amount (fig. 5). In Weigert-Pal preparations there was almost complete demyelination of the white fibres. Only a minimum of faintly stained narrow shrivelled fibrils and an occasional one rather thicker, with varicose swellings like the bulbs of Higginson's syringes, were stained. A somewhat similar picture was obtained with Sudan III in frozen sections, but there was no result on staining for fat by the Marchi method.

Cerebellum.—The cortex was very atrophic. The granular layer was much thinned, being in most places only two or three cells thick. The Purkinje cells were also much diminished in number and irregular in their distribution, with great diminution in their cortical arborisations, some of the primary divisions or 'antlers' of which had enormous globoid or irregular swellings upon them as described by Schob in 1912 (fig. 6), who pointed out their fatty nature. In the white matter there was marked demyelination, especially in the smaller folia, and this change, though still marked in degree, was rather less in and around the dentate nuclei (fig. 7).

Pons.—A transverse section at the level of the nuclei of the trigeminal nerve showed well-marked demyelination, especially of the pyramidal bundles; a transverse section through the medulla oblongata about the middle of the olivary region also showed demyelination of the pyramids and anterior spinocerebellar bundles, while the medial lemnisci, etc., were not similarly affected. In the pons and medulla Marchi's stain gave no evidence of any recent fatty change, only an occasional myelin sheath being stained in the least demyelinated areas. In some parts of the pons the vertical, and especially the pyramidal, and in others the horizontal fibres, were more affected. All stages of degeneration were seen from moderate to complete loss of myelin, some fibres showing varicosity, others attenuation, etc.

Optic Nerves.—Here there was no recent fatty change in the myelin sheaths demonstrable with osmic acid, though small fatty granules were

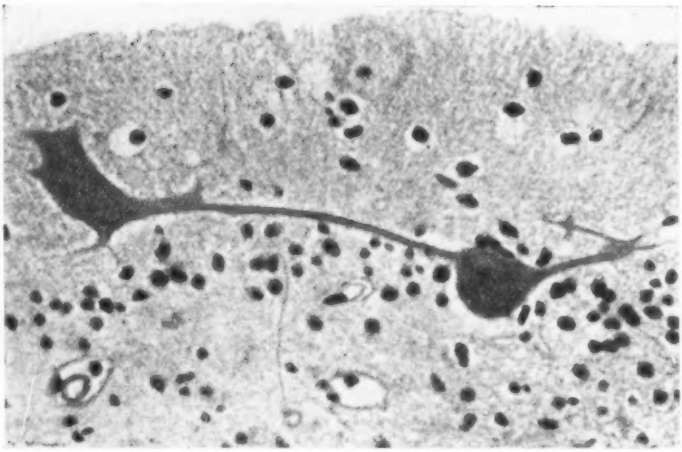


FIG. 6.—Cerebellar cortex showing extreme atrophy, diminution of the cells of the graular layer, and one of the malformed Purkinje cells with an enormous swelling upon one of its 'antlers.' (Hæmatoxylin-eosin.)

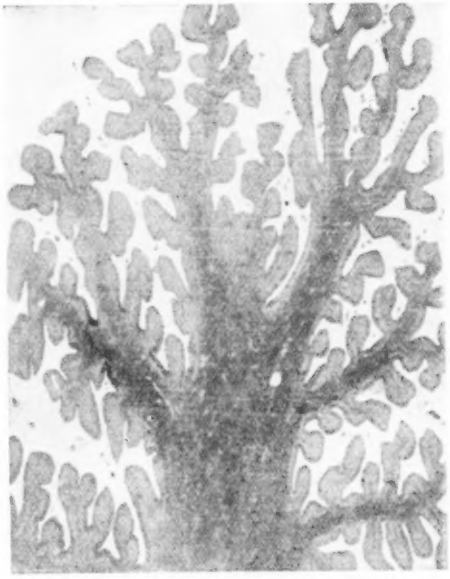


FIG. 7.—Cerebellum. Low-power section showing the atrophy of both cortex and medulla. (Hæmatoxylin-eosin.)

visible in the cytoplasm of endo- and perithelial cells of the small vessels. The myelin sheaths themselves were variable in appearance (fig. 8), some appearing varicose or beaded, others attenuated and in process of disappearing.

The condition of the axis-cylinder processes, etc.—In a series of frozen sections stained by the Bielschowsky method the neurofibrils of the dendrites and axis-cylinder processes of the nerve-cells of the cerebral cortex generally appear to be markedly reduced in number and complexity, though surviving in greater degree than might have been expected from a study of sections

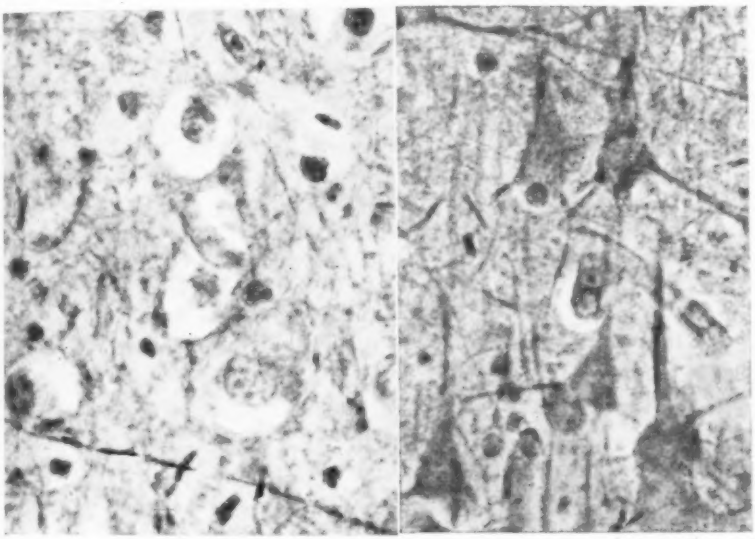
FIG. 8.—High-power view of optic nerves showing beading and attenuation of myelin sheaths and partial atrophy. (Wiegert-Pal).

stained by other methods (fig. 9). Within the nerve-cells they traverse only the periphery of the cytoplasm and appear to be pushed aside by the globose swellings. In many of the cells, however, they have entirely disappeared. In the white matter the axis-cylinders also survive in much greater degree than the myelin sheaths, though they are distinctly scantier in the white matter of the cerebellum than in that of the cerebrum.

The spleen was unfortunately not kept for section, but the liver did not show any appreciable increase in the reticulo-endothelial Kupffer cells in the walls of the intercellular blood-sinusoids, and one may therefore infer that the condition was not of the Niemann-Pick splenohepatomegalic type.

SUMMARY.

A case of socalled cerebromacular degeneration is recorded in which, however, macular changes were not in evidence. The patient, a male child of non-Jewish extraction, was three years old at the onset of the disease, which lasted three years, with progressive loss of vision, hearing, and mentality. The pathological findings were those typical of this group of diseases, though perhaps more advanced and extreme than in some of the cases recorded—probably owing to the long course of the illness. All the



Pyramidal cortex of case. Pyramidal cortex of normal control.

FIG. 9.—Thick frozen sections stained by Bielschowsky's method for neurofibrils. It was difficult to obtain these in focus in any numbers in the photographs, which are shown here to illustrate loss of shape and character of the nerve-cells, which are scarcely recognisable in the cerebromacular case.

nerve-cells throughout the brain and cord showed varying degrees of globose swelling, the presence of fatty granules in the surviving perinuclear cytoplasm, disappearance of Nissl-granules, and a gradual but progressive diminution of dendrites. Some of the latter had curious swellings upon them (e.g., on those of the Purkinje cells). Progressive and widespread demyelination of the white nerve-fibres of the cerebrum (including the optic nerves), cerebellum and brainstem was found. In the latter the pyramidal bundles were especially affected. In the spinal cord, although the nerve-cell changes were well marked, the white matter was relatively little affected,

This case falls clinically into the group described by Batten and named by him ‘ cerebromacular degeneration.’ A thorough pathological investigation revealed no evidence in support of the theories of Sachs, Bielschowsky and others that the disease is related to Gaucher's and Niemann-Pick's disease and is a form of general lipoid degeneration. Schaffer's view that cerebromacular degeneration is a primary degeneration of the ganglion-cells seems to gain support from this case.

THE CEREBROSPINAL FLUID IN 230 CASES OF GENERAL PARALYSIS AFTER MALARIAL TREATMENT.

By

B. REID, Whittingham County Mental Hospital.

Among modern methods of diagnosis of late syphilis of the nervous system, examination of the cerebrospinal fluid has proved of very great value. At this hospital special interest has centred round cases of general paralysis, and recently the problem of changes before and after treatment with malarial inoculation has been investigated. This has been done by a number of other writers, but we have been specially favoured in this study by the fact that malarial treatment was first started in England at Whittingham Mental Hospital in July, 1922. The records therefore extend over a period of nine years and include a series of 230 cases treated and examined during that period. In addition I have been able to collect cerebrospinal fluid for examination from 14 patients who were discharged as recovered and who are now living at their own homes. More patients living at home might have been induced to allow lumbar puncture, but minor after-effects were sometimes complained of and on one occasion a patient collapsed merely from the pin-prick in the skin. These 14 cases, however, are of value in that the patients had been discharged for varying periods of time; they form a sufficient basis on which to formulate reasonably definite views. The purpose of this article is to ascertain the results in the cerebrospinal fluid after malarial treatment of general paralysis among three distinct classes of patients:

1. Patients not recovered and now dead.
2. Patients not recovered and now living in hospital.
3. Patients recovered and now living at home.

The changes which occur in the cerebrospinal fluid and the tests applied are too well known to require detailed description here. They may however be briefly mentioned with special reference to the tests carried out on the cases investigated in this hospital. Cell counts are increased. The Ross-Jones and Pandy tests show an increase of globulin. The Wassermann test of the cerebrospinal fluid is positive, and the Lange colloidal gold reaction gives a zone I curve. In addition to these tests the colloidal gamboge reaction has been carried out in the cerebrospinal fluid and an experience of

three to four thousand examinations in this hospital shows it to be as reliab as the colloidal gold test. All tests were performed in the hospital laborato with the exception of the Wassermann which was done in Mancheste University laboratory.

GENERAL RESULTS

A detailed presentation of 230 cerebrospinal fluid examinations with changes recorded after treatment is a difficult task on account of the large numbers involved, but it is possible to give the general findings after careful consideration. The first point is that in nearly all cases examined within one or two months after treatment with malaria, the cerebrospinal fluid shows a decrease in the cell count, very often in a marked degree. The diminution in the cells present remains even when no other improvement in the laboratory examinations can be found at later times. This finding is not without significance and suggests that the pathological condition of the brain and meninges producing the increased number of cells in the fluid has been arrested, even at such an early time after treatment. To begin with, this is the only change found from that of the typical cerebrospinal fluid of the general paralytic. As years go on, however, further changes appear. There is a gradual return of the fluid to a normal condition after treatment with malaria. This statement is correct for the majority of cases, but a small number, even after five or six years, still yield to laboratory examination findings of general paralysis.

Discussing each test individually one finds that the Pandy and Ross-Jones tests for increased globulin both gradually return to normal and so give a negative result. The Wassermann test of the fluid proves more resistant to change than any of the other laboratory tests, and even after all other findings are negative, a positive Wassermann may still be recorded. The Wassermann, however, also shows a gradual return to negative, but several years elapse in most cases before this change appears. The colloidal gold test and the colloidal gamboge test show changes which are more or less similar. In a certain number of cases they change to a zone II curve and if taken a year or so later this zone II curve may be found to have disappeared and a negative response may be found. Most of the other cases, after a year or more, give a curve which is only slightly changed from the normal or negative reaction. Thus it may be said that in examining a large number of fluids after treatment with malaria, there is, over a period of several years, a gradual return to negative reactions in many; that many more show a diminution in the pathological findings; and that only a small number still give all the reactions of general paralysis several years after treatment.

No attempt can be made to differentiate between the changes in the recorded cases and in those which have not improved mentally. It appears

rom the examinations that there is no appreciably quicker improvement in he fluid among those who fare well than among those who do not.

Again, as regards the period of time for a positive reaction to become negative or nearly negative no definite duration can be fixed. Some may do so in one year while others take two, three, four or more years. The average period of time would appear to be about two to three years.

The findings in the 14 who had fluid withdrawn at their own homes and who were in a satisfactory mental condition after treatment are as follows:

	Ross-Jones test	Pandy test	Cell count	Wassermann reaction	Colloidal gold reaction	Colloidal gamboge reaction
1	neg.	neg.	1·6	+	0011000000	100000
2	neg.	neg.	6·0	doubtful	0121000000	100000
3	+	+	16·6	+	0122000000	200000
4	neg.	neg.	2·6	neg.	0011000000	000000
5	+	+	2·6	+	0011000000	000000
6	+	+	2·3	+++	0122000000	100000
7	neg.	neg.	0·6	neg.	0011000000	000000
8	neg.	neg.	0·6	neg.	0011000000	000000
9	neg.	neg.	0·3	+++	0011000000	000000
10	+	+	3·0	neg.	5554210000	210000
11	+	+	4·0	neg.	5543200000	221000
12	+	+	2·0	doubtful	0022100000	221000
13	+	+	5·0	+++	5554320000	221000
14	+	+	5·3	weak +	1122000000	100000

In most of these cases the approach to a normal finding is definite, and some are completely negative.

It has been my experience throughout to find that mental improvement precedes marked improvement in the spinal fluid, but that an approach to a normal fluid gradually makes its appearance. This progress to normality is found to an equal degree in the patients who still remain in hospital and whose mental state shows no improvement. It thus appears that following treatment of general paralysis with malaria, there is, in the majority of patients, a diminution of pathological changes as found by laboratory tests, and that over a period of several years there is a gradual return to a normal fluid. This improvement in the fluid is therefore no index to the mental improvement in the patient. Patients who remain demented may after the lapse of several years give a negative finding in the fluid.

These findings seem to be of definite help in assessing the value of malarial treatment and indicate that such treatment has the power of bringing about a return to normal conditions in the central nervous system.

As further confirmation of this return to normality, whereas before treatment with malaria the presence of spirochætes in the brain was of frequent occurrence in general paralytics, we can no longer demonstrate the organisms of syphilis in the brain employing the same methods as formerly. Thus both from the point of view of mental improvement and also of the laboratory findings, malarial treatment can be advocated as of definite value in general paralysis and the earlier such treatment is given the better the results will be.

CONCLUSIONS.

1. The cerebrospinal fluid improves in patients suffering from general paralysis after treatment with malaria and may become normal. This improvement usually takes several years to occur.

2. The improvement in the fluid occurs in patients who have improved mentally and also in those who do not improve.

Editorial.

PROBLEMS OF DISSEMINATED SCLEROSIS.

DURING the last decade disseminated sclerosis has undoubtedly occurred with increasing frequency, and at the present time few neurologists would challenge the statement that in this country it is now the commonest organic disease of the nervous system, not even excluding neurosyphilis. Although its etiology still remains obscure and but little progress has been made in either its prevention or treatment, some advance is apparent towards a better understanding of the clinical aspects of the disease. Until comparatively recently it was taught that disseminated sclerosis made its appearance insidiously, in some form of slowly progressive paralysis, whereas now it is generally agreed that the onset is almost invariably sudden, that is, as regards the appearance of the first symptom. This may consist in weakness or paralysis of one or more limbs, the development of paræsthesia, vertigo, or of an ocular palsy, or the sudden occurrence of amblyopia. The latter symptom is due to an acute retrobulbar neuritis; and it is remarkable that even at the present time many practitioners, not excluding some ophthalmologists, are not fully alive to the fact that an acute unilateral retrobulbar neuritis, without other symptoms or signs, is usually the initial manifestation of what later will prove to be disseminated sclerosis. Unilateral retrobulbar neuritis is probably far more frequently the first sign of the disease than is generally supposed. Upon taking the history of a case of manifest disseminated sclerosis, the patient not infrequently omits to mention that some years before he suffered from a temporary loss of vision in one eye, merely because he does not connect such an occurrence with his present condition; and this important point in the history is therefore missed unless special inquiry is made. Again, many patients are met with, subsequently proved to be suffering from disseminated sclerosis, in whom all teeth have been extracted,

sinuses explored and even appendices removed for an earlier retrobulbar neuritis which at the time was attributed to some 'toxic' influence. The frequency of acute unilateral retrobulbar neuritis as an early symptom was emphasized by Dr. W. J. Adie in opening a discussion on the symptomatology of disseminated sclerosis at the last annual meeting of the British Medical Association. In Dr. Adie's series of 118 cases, acute retrobulbar neuritis had occurred in 41. It may appear many years before other signs of the disease are apparent; in recorded cases the interval varies from a few weeks to over twenty years. In fact, with increasing experience the more is one inclined to regard every case of acute unilateral retrobulbar neuritis as likely to prove an example of early disseminated sclerosis.

Simultaneous and early consecutive affection of both eyes is admittedly rare; and it is a matter yet to be determined whether such cases are indeed examples of disseminated sclerosis or of some other condition such as acute disseminated encephalomyelitis. This raises another important problem still unsettled: is acute disseminated encephalomyelitis a separate clinical entity or is it merely an acute form of disseminated sclerosis? Neurological opinion is divided. Some authorities favour the view that the two conditions, in spite of their resemblances, are distinct diseases, while others regard their identity as proved by the similarities of the histological lesions. Some consider that certain clinical features such as the occurrence of pyrexia, loss of thermal sensibility, and the presence of pain and a normal cerebrospinal fluid are usually found only in disseminated encephalomyelitis and serve to distinguish this condition from acute disseminated sclerosis. Also, the former may occur in young children while the latter is rare before the age of 16 years. Loss of vision is rare in encephalomyelitis, but when present both eyes are usually affected. The most important point, however, is that in cases of acute disseminated encephalomyelitis, when the initial phase passes off sometimes with complete recovery, no further relapse will occur. On the other hand, remissions extending over many years are by no means infrequent in disseminated sclerosis. Consequently it would seem that the only method of definitely settling the problem is for a number of cases, presumed to have recovered from acute disseminated encephalomyelitis, to be followed up for a number of years.

Several instances of familial disseminated sclerosis have been recorded, but their distinction from some of the more unusual forms of heredofamilial ataxia is admittedly difficult. Both diseases are protean in their manifestations and have many physical signs in common. It would appear that three features only—which occur in disseminated sclerosis and not in heredofamilial ataxia—can be relied on to distinguish one disease from the other. These are (1) the presence at some time during the course of an acute retrobulbar neuritis; (2) the history of definite remissions; and (3) if present, the occurrence of a Lange curve of the paretic type in the cerebrospinal fluid.

A further aspect of disseminated sclerosis to which little attention has been paid is the possible occurrence of transient mental disturbance as an initial symptom. The disturbance is usually that of confusion or of hypomania, and it may occur at a time when no definite physical signs of organic disease are apparent.

An additional help in the early diagnosis of the disease is afforded by taking a speech record according to the method of E. W. Scripture. The tracing shows an ataxia of the vocal muscles long before any disturbance of speech is apparent to the ear. This ataxia can also be shown by means of sound tracks, and it appears to be constant in all cases excepting a few with a purely spinal cord localization.

Examination of the cerebrospinal fluid is playing an increasingly prominent part in the diagnosis of disseminated sclerosis. Certain findings are of much significance, viz. cell count (lymphocytes) normal or only slightly increased; protein content 0·04 per cent. or less; globulin in slight excess; Lange curve of the paretic type with a negative Wassermann reaction. A paretic Lange reaction is frequent at the onset, during acute relapses and not infrequent in uncomplicated acute retrobulbar neuritis. During remissions the Lange curve may be luetic in type or negative. Further experiences will probably show that a positive Lange reaction can be obtained at some time during the course in all cases.

In spite of much investigation the etiological factor and even the pathological nature of disseminated sclerosis still remain in doubt. Most authorities favour an inflammatory reaction rather than an abiotrophy. The method of treatment, however, inspired by possible belief in a spirochæte as the causative agent, remains the most popular, that is, the injection

of one of the arsenobenzol compounds such as novarsenobillon or silversalvarsan. Arsenic, in the opinion of many, appears to have some inhibitory influence on the progress of the disease. In such a malady, characterized by remissions of variable duration, the value of any treatment is notoriously difficult to assess, and it must be admitted that any retarding action of arsenic is only slight. Cacodylate of soda, antimony, iodine and mercurochrome must by now have been discarded by all, but bismuth is still used by some. Non-specific protein therapy in the form of courses of T.A.B. vaccine or ' pyrifer ' given intravenously often results in temporary improvement. It would seem that the ' vaccine ' made from the alleged ' spherula insularis ' must be regarded only as a form of non-specific protein therapy. The results of feeding with liver and liver extracts—based most probably on a false etiology—are by no means impressive. Treatment is likely to remain unsatisfactory until more is known regarding the true etiology and pathology of the disease.

Abstracts.

Neurology.

NEURO-ANATOMY AND NEUROPHYSIOLOGY.

[66] **The cranial nerve roots** (Le radici nervose encefaliche).—G. L. Francesconi. *Riv. di pat. nerv. e ment.*, 1932, xxxix, 415.

The author here attempts a new classification of cranial nerve roots, distinguishing 24 according to the nature of their function. This arrangement retains the unity of the roots corresponding to the cranial segments. They are described from the point of origin to the point of distribution without reference to the fact that they may for part of their course be joined up with another root. The exact details of this arrangement must be studied in the original article, as it is impossible to abstract the whole of this careful and elaborate work in any reasonable space. It may be useful, however, to give a list of the roots.

A. Roots concerned with the relation of the individual to the environment.
   a. Motor.
      1. Common oculomotor.
      2. Trochlear oculomotor.
      3. Abducens oculomotor.
      4. Motor to the muscles of mastication.
      5. Facial.
      6. Glossopharyngeal motor to body muscles.
      7. Pneumogastric motor to body muscles.
      8. Accessory.
      9. Hypoglossal.
      10. Spinal.
   b. Of general sensibility.
      11. Trigeminal.
      12. Glossopharyngeal (bodily sensation).
      13. Intermediary sensory root of Wrisberg-Sapolini.
   c. Of special sense.
      14. Olfactory.
      15. Visual.

16. Auditory.
17. Taste.
18. Equilibrium.

B. Roots concerned with the vegetative life.

a. Motor.

19. Common oculomotor intrinsic muscles.
20. Pneumogastric visceral motility.

b. Sensory.

21. Pneumogastric visceral sensibility.

c. Secretory.

22. Lachrymal.
23. Superior salivary.
24. Inferior salivary.

R. G. G.

[67] **Corticofugal and association fibres arising from the cortex of the vermis of the cerebellum.**—L. BENDER. *Arch. of Neurol. and Psychiat.*, 1932, xxviii, 1.

THIS is a report of a study of the secondary degeneration of the nerve-fibres resulting from lesions in the cortex of the cerebellar vermis.

It was possible to trace short cerebellofugal fibres from the cortex to the central nuclei, long cerebellofugal fibres in four tracts to the various parts of the brainstem, and intracerebellar association fibres to other parts of the palæocerebellum.

Short corticofugal fibres from the superior part of the vermis pass mainly to the roof nuclei. From the inferior part they pass mainly to the globoid nuclei, and there is also a definite well-formed tract to the phylogenetically oldest part of the dentate nucleus.

Long corticofugal fibres from the vermis may be classified as follows:

Fibres that enter the brachium conjunctivum to end in the opposite red nucleus or in the mesencephalic root of the fifth nerve.

Perforating fibres that pass directly from the cortex to the vestibular nuclei, reticular formation, grey matter accompanying the posterior longitudinal bundle and descending tract of Deiters' nucleus.

Fibres that arise from the most anterior part of the vermis (especially from the lingula) and take a position between the central nuclei and ventricle where some of them decussate. Both crossed and uncrossed fibres enter the hooked tract of Russel and end in all of the vestibular nuclei and related grey matter, as do those of the second group. They follow especially the spinal vestibular nucleus and end in it at all levels. Some of these also follow the course of external arcuate fibres. To this group the name ' bundle of the ventral decussation ' is given.

Fibres of similar origin that take a position dorsal to the central nuclei where they form a partial decussation. From here they pass to the lateral surface of the restiform body, where they ascend and descend and course as external arcuate fibres to reach the nuclei of the raphe at all levels and possibly also the olives. This is the ' bundle of the dorsal decussation.'

Intracerebellar association fibres connect all parts of the palæocerebellum (vermis and flocculi), but these are least numerous in the oldest parts and most abundant in the phylogenetically younger, intermediate (i.e., the lobulus medius medianus of Ingvar).

R. M. S.

[68] **The cortical centres of the labyrinth.**—E. Spiegel. *Jour. Nerv. Ment. Dis.*, 1932, lxxv, 504.

Experiments on the subject were done on dogs and cats in which different circumscribed cortical areas were poisoned by means of applying strychnine, and then the effect of labyrinthine impulses were studied on these animals. The experiments prove that labyrinth stimulation produces epileptiform attacks (starting in the muscles of the eyelid and the ear) if the posterior parts of the gyrus ecto- and suprasylvius are made sufficiently hypersensitive by strychnine poisoning. In such cases loud noises could produce similar attacks, as described by Clementi. Painful stimulation or violent concussion by repeated striking on the operation board with a hammer did not produce such attacks. After concussion only slight general reflex contractions were observed; it may therefore be concluded that epileptiform convulsions after rotation are caused by simultaneous concussion of the animal instead of the irritation of the labyrinth.

Sometimes poisoning of the dorsal part of the gyrus ectosylvius (Nos. 11, 12, 19) was sufficient to produce such spasms after stimulation of the labyrinth; in most cases, however, the effect was evident only after strychninization of the neighbouring gyri (first, third posterior arcuate gyrus). The experiment was mostly positive when the gyrus sylvius and gyrus suprasylvius posterior were poisoned in their dorsal part.

A further study concerned the question whether removal of the cerebellum would prevent the production of epileptiform attacks by rotation after application of strychnine to the cortex. In spite of almost total extirpation of the cerebellum it was possible to produce convulsions by stimulation of the labyrinth which issued from the ear muscles on the side opposite to the poisoned cortex.

Although labyrinthine impulses may be conducted to the cortex partly over the cerebellum, yet it is by no means necessary that the cerebellar condition should be intact in order to allow the labyrinthine impulses to enter the cerebrum. This is also in conformity with a clinical observation

made by Fischner and Pötzl, who produced labyrinthine vertigo in a patient also after resection of one cerebellar hemisphere.

Further investigations will have to answer the question how far these tracts are commonly used by the labyrinthine impulse on its way to the cortex. The more exact limits of the cortical areas receiving labyrinthine impulses, especially in higher mammals, will likewise require study. The labyrinth is represented in the cortex and its cortical localization is to be sought in a part of the temporal lobe. If this region is hyperexcitable, it is possible to produce epileptiform attacks by irritation of the labyrinth.

R. G. G.

[69] **Cerebral circulation. Afferent impulses from the blood-vessels of the pia.**—M. Levine and H. G. Wolff. *Arch. of Neurol. and Psychiat.*, 1932, xxviii, 140.

The authors have attempted to demonstrate physiologically that the nerve-supply to the vessels of the pia mater (which has been proved beyond question) possesses sensory as well as motor functions.

Bulbocapnine administration provided an adequate method by which cats under local anæsthesia could be immobilized without interfering with the galvanic skin responses.

Galvanic skin responses were obtained by faradic stimulation on the pial blood-vessels and on the relatively nonvascular pia over the cerebral cortex.

Stimulation on the pial vessels produced galvanic skin responses more than twice as often as did stimulation on the relatively nonvascular pia. Further, stimulation on the vessels produced galvanic skin responses which were much larger than those that resulted from stimulation on the relatively nonvascular pia.

It was concluded that the impulses produced by the stimulation were afferent rather than efferent; that these afferent impulses had arisen in blood vessels and pia rather than in the underlying cortex; that the responses had more than twice the frequency and magnitude when the afferent impulses arose from blood-vessels than when they arose from the relatively nonvascular pia, and therefore, that the pial blood-vessels possess an afferent as well as an efferent nerve-supply.

Afferent impulses from the pial blood-vessels may be of importance physiologically in the mechanisms involved in the maintenance of a constant internal environment through the regulation of the cerebral circulation, as well as pathologically in the production of certain headaches and convulsions.

R. M. S.

[70] **Studies in sensation. III. Chemical factor in the stimulation of end-organs giving temperature sensations.**—H. C. Bazett and B. McGlone. *Arch. of Neurol. and Psychiat.*, 1932, xxviii, 71.

Measurements have been made of the temperature changes in the forearm, hand or fingers on release of stasis, both with the limb in air and immersed in water at varying temperature, and the values obtained have been compared with the sensations induced so as to test Ebbecke's hypothesis of the origin of temperature sensations, which assumes that the appreciation of both warmth and cold depends on thermal gradients of varying intensity set up at different depths as warm blood enters cooled skin or vice versâ.

Sensations of intense warmth or heat were found to be induced on release of stasis in a limb maintained before, during and after stasis in a bath at the blood temperature level, so that on release no changes in temperature occurred, and no thermal gradient was established. These sensations were found to vary roughly according to the probable acidity of blood flowing into the dermal area, to be more intense with long periods of stasis than with shorter, and greater with stasis of the whole forearm than with that of a finger only. They are considered to be elicited by a chemical stimulus (acting probably through the establishment of a chemical gradient) derived from metabolic processes, particularly in muscle tissue, and by a substance that varies in concentration both during asphyxia and as the result of temperature changes in a manner similar to that of acid. The possibility of acid itself being the stimulus is discussed but is considered somewhat improbable.

The sensations at other temperatures are considered as caused by chemical changes of the same type antagonized to a greater or less extent by opposite chemical changes induced by thermal differences.

Some data are presented of the thermal changes and gradients in the dermis associated with temperature sensations induced by varying the bath temperature. They are considered to be most readily explained by some modification of Ebbecke's hypothesis.

Tingling sensations occurring during stasis and on its release are shown to originate peripherally in the area exposed to stasis and to be modified by the temperature of this part; they are not dependent on pressure on the nerve-trunks.

R. M. S.

[71] **Nerve degeneration in poliomyelitis.**—P. Heinbecker, G. H. Bishop, and J. L. O'Leary. *Arch. of Neurol. and Psychiat.*, 1932, xxvii, 1402.

The vagus, cervical sympathetic and various somatic nerves were severed aseptically in fifteen young adult cats and one monkey. The animals were

killed at intervals from twelve to one hundred and seventeen hours and the distal segments examined by physiological and histological methods. For the functional study, the cathode-ray oscillograph was employed to record the conducted action potentials of the severed nerves and their controls. For microscopic examination, chief reliance was placed on the appearance of fragmentation in the medullary sheaths, as evidenced in osmic acid-stained preparations. Ranson's pyridine-silver modification of the Cajal method was used to check the relative rates of degeneration in myelinated and nonmyelinated fibres.

Results of the physiological study showed that increase in the absolutely refractory period may be detected as early as twelve hours after section. Significant reduction in potential area was usually not apparent before thirty-six hours. The fibre groups of somatic and visceral nerves characterized by different physiological properties lose their action potentials in sequence. The potential from visceral afferents disappears first at from fifty to seventy hours; the potential from somatic motor and sensory fibres, five to ten hours later; then the potential from myelinated autonomics, and finally, the potential from unmyelinated fibres. A potential from unmyelinated fibres has not been elicited later than ninety hours postoperatively.

In differential counts of three groups of myelinated fibres (large fibres, selected range, diameter from 6·7 to 18·1 microns, sheath thickness, from 1·4 to 2·4 microns; medium-sized, selected range, diameter from 4·8 to 6·5 microns, sheath thickness, from 0·43 to 0·85 micron; fine, selected range, diameter from 1·4 to 3·0 microns, sheath thickness, from 0·4 to 0·65 micron), fragmentation was set as an arbitrary criterion of degeneration. Those fibres not exhibiting fragmentation in the portion available for study in a 5-micron longitudinal section, stained with osmic acid, were counted in one group; those exhibiting fragmentation in at least one place, in another. The comparative values indicate that fragmentation occurs earlier on the average in the medium-sized, relatively thin-sheathed group, next in the large, myelinated fibres, and finally in the smallest, myelinated ones. Numerous nonfragmented unmyelinated axons persist at a stage when in osmic acid stained preparations the sheaths of almost all myelinated fibres appear to be fragmented.

R. M. S.

NEUROPATHOLOGY.

[72] **The histopathology and pathogenesis of poliomyelitis** (Zur Histopathologie und Pathogenese der Poliomyelitis).—W. Spielmeyer. *Zeits. f. d. g. Neurol. u. Psychiat.*, 1932, cxli, 159.

In contrast with what obtains in other cases of meningoencephalitis, the lesions of poliomyelitis in the brain first cause infiltration of the

central substance and move outwards; the meninges are involved from the cerebral side. Focal manifestations may be altogether lacking although vascular infiltrates are present. The lesions of the vascular system in the neuraxis do not help to solve the problem of local affinity. They are the expression of a resorptive 'inflammation,' a distant reaction to primary inflammation of grey matter. The focal lesions are not clustered round vessels invariably or often. Since the brain lesions are those of an encephalomeningitis, it is clear the fluid does not convey infection.

In the motor zone of the cortex, no systematization of the lesions can be traced; the virus has no selective action on motor ganglion-cells, nor is neuronophagia or cell-outfall so marked as in the case of the cord. In the latter, again, any part whatsoever of the grey matter may be affected, with the exception, perhaps, of the substantia gelatinosa and the tip of the dorsal horns. Speaking broadly, the anterior two-thirds of the grey matter contains the bulk of the lesions. Within that area, wide variations occur. Dorsal horns and Clarke's columns may be more diseased than any other section. Spinal root-ganglia, gasserian ganglion, and the nuclei of the cerebellum may be involved. Many motor nuclei, on the contrary, are often spared. In other words, poliomyelitis is no disease of the motor system, and attacks mechanisms whose functions are quite diverse. The virus also damages substantia nigra, globus pallidus, thalamus, etc.

The only permissible conclusion is, that neither the vasal factor nor the system factor accounts for the histopathological phenomena. Local vulnerability, that is to say, has not yet received a convincing explanation. This affinity of the virus for certain masses of grey substance is independent of the spot at which the virus enters, or is made to enter, the body—an old observation, though some writers are under the impression it is novel. No theory has as yet accounted for the observed fact that the anterior two-thirds of the spinal grey matter harbour the maximum number of foci.

As is known, evidence is accumulating which goes to show that the virus travels to the spots it favours by means of axonal spread.

The paper finishes with a discussion of the reasons for the differences of the histological reactions in different places and alludes to the occasional occurrence of lesions in white matter.

S. A. K. W.

[73] **Histological studies of the brain in cases of fatal injury to the head.**—C. W. Rand and C. B. Courville. *Arch. of Neurol. and Psychiat.*, 1932, xxvii, 1342.

In this contribution the authors have been concerned with the changes in classic neuroglia as the result of injury to the human brain. In the first few days following trauma, the astrocytes in the immediately adjacent area

undergo regressive changes with the formation of amœboid glia. In the zone nearest the point of injury the cells undergo complete destruction. Beyond this zone of destruction active proliferation takes place. Here, within a few hours after injury, double nucleated forms indicate active direct cell-division. In a rather narrow intervening zone, apolar, bipolar and unipolar forms have been found, which, because of their ready impregnation with gold sublimate, are probably viable cells. This is furthermore suggested by the presence in these cells of double nuclei having normal morphological characteristics. These elements are interpreted as regressive forms capable of reversible action.

Gliosis occurs only as a result of tissue destruction. The degenerating tissue probably serves as a chemical stimulant to the regional astrocytes. In the formation of the glial scar, glia-cells assume their active or kinetic function of space compensation in the process of healing. Extensively damaged brain tissue, which requires a rather prolonged activity on the part of the phagocytic mesoglia, delays for a considerable period the formation of the ultimate glial scar. The astrocytes taking part in this delayed reaction are themselves often victims of regressive change, particularly of a hyaline nature.

Judging from studies on comparatively recent cases, it seems likely that the neuroglial reaction is a purely local affair and consequently that generalized gliosis following head injury does not occur. The remote clinical sequelæ of such injuries are largely to be interpreted on the basis of focal and distant cortical injuries. In addition, there are numerous interruptions in the connecting pathways incident to focal and subcortical injury, together with the changes following widespread petechial hæmorrhages. In this sense localized gliosis is only a histological manifestation of the damage which produces the symptoms.

R. M. S.

[74] **The etiology of disseminated sclerosis** (Neues zur Ätiologie der multiplen Sklerose).—G. Steiner. *Der Nervenarzt*, 1932, v, 281.

In 1928 the author found spirochætes for the first time in the brain of a case of disseminated sclerosis. By September, 1930, the number of ' positive ' cases has risen to 7 out of 24. A description of his findings was given before the Neurological Congress of Berne last year, and slides of the ' spirochæta myelophthora ' shown. In addition, Dr. Steiner drew attention to the occurrence of ' silver cells ' in the periadventitial lymph-spaces in the same affection, cells which appeared to be laden with what might be considered spirochætal debris. Similar argyrophil inclusions were to be seen in round cells in the parenchyma, in the neighbourhood of infiltrated vessels.

The present communication deals with the same questions and is illustrated with a number of microphotographs. A new case has proved to be richer in spirochætes than its predecessors; and by means of a Zeiss multiplicator it has been possible to photograph in series, at different optical levels, the objects under discussion. From the pictures there cannot be any doubt of their spirochætal nature. The fresher and more acute the lesion, the more readily may spirochætes be expected therein; chronic and organized lesions seem to be free from them.

This communication deserves close attention on the part of the neurologist and is conspicuous for the temperate and judicial character of the author's comments on his results.

S. A. K. W.

[75] **Research on the microglia and oligodendroglia** (Ricerche sulla microglia e la oligodendroglia).—D. Bolsi. *Riv. di pat. nerv. e ment.*, 1931, xxxvii, 1.

The author shows that the microglia under the influence of certain toxic factors may show acute swelling which Penfield and Cone thought was a specific reaction of the oligodendroglia; there is however no evidence of any true similarity in structure or function of the two types of cells, which much research has proved to be fundamentally different. He further describes his own method of vital colourization, which confirms him in his opinion that the microglia should be classed in the reticulo-endothelial system.

R. G. G.

[76] **Hemispheric cerebral sclerosis secondary to meningoencephalitis and associated with multiple visceral blastomata** (Sclerosi cerebrale emisferica da pregressa meningoencefalite associata a blastomi viscerali multipli).—P. Ottonello. *Rass. di studi psichiat.*, 1932, xxi, 1.

The interest of this case consists in the association of multiple tumours with a unilateral cerebral sclerosis secondary to a postnatal meningoencephalitis. The assertion is commonly made that tuberous sclerosis alone is apt to show this connexion, but according to the history and description of this case the statement must be reconsidered. The tumour lesions consisted of a hypernephroma of the kidney, lipoma of the diaphragm, and fibromyoma of the stomach. The brain lesions were clearly those of a postnatal inflammatory process involving both cortex and meninges on one side, with resulting atrophy of the hemisphere.

S. A. K. W.

[77] **The sympathetic paths in the medulla** (Étude sur les syndromes hémibulbaires: les voies sympathiques dans le bulbe).—Knud Winther. *Acta Psychiat. et Neurol.*, 1932, vii, 719.

The occurrence of paralysis of the cervical sympathetic in cases of unilateral lesion of the medulla is familiar to the neurologist, but the exact position of the 'centre' has not been located satisfactorily. Cases of thrombosis of the posterior inferior cerebellar artery can be utilized for the purpose, though the area involved is apt to be rather too large and the syndrome too complicated. Dr. Winther has had the opportunity of studying pathologically four cases in point, and comes to the conclusion that the enophthalmus of the syndrome is not caused by paralysis of the orbital muscle of Müller but is due to atrophy of orbital fat. The sympathetic paths concerned are situated in the lateral part of the formatio reticularis, between the nucleus ambiguous and the descending root of the fifth. The ocular fibres are close to the former; those for the face are rather in front of the others. Those for the body are still farther forward, in relation with the spinothalamic tract.

S. A. K. W.

[78] **The Kahn test applied to spinal fluids.**—M. B. Kurtz and N. W. Larkum. *Amer. Jour. Syphilis*, 1932, xvi, 377.

The results obtained with a modified Kahn test on the cerebrospinal fluid are detailed. In order to achieve greater sensitivity, concentrations of 20 in 1, as well as the usual 10 in 1, were used and the fluids were also tested with 'presumptive' Kahn antigen (see this Journal, vol. x, p. 336). Special precautions were taken to avoid an excess of ammonium sulphate in the concentrated globulin solutions. By these improvements the authors claim to have increased the number of syphilitic fluids which give a positive Kahn test. They place no reliance however on the 'presumptive' antigen technique, which is only used as a 'check on the general technique of the test.' But they consider that unless concentrations of at least 15 in 1 are tested with standard antigen the result should be returned as an incomplete test.

J. G. G.

[79] **The posterior fossa in epilepsy.**—G. W. Swift. *Jour. Nerv. Ment. Dis.*, 1931, lxxiv, 689.

This anatomical study seems to establish the following facts.

1. The presence of anomalous sinuses and asymmetrical development of the transverse and sigmoid sinuses are common in epilepsy.

2. The occipital bone in epileptics is frequently smaller than normal, resulting in a small posterior fossa.

3. The accumulation of cerebrospinal fluid occurs first over the cerebral hemispheres, beginning in the frontal and extending backward, and later resulting in dilatation of the ventricles.

4. Encephalograms should be taken routinely in epilepsies to reveal changes in the venous sinuses, the size of the occipital fossa and the distribution of cerebrospinal fluid.

With these facts established, an effort is being made to ascertain whether by operative procedure it is possible to relieve the pressure against the sigmoid sinus and thus stimulate the return venous blood from the brain. In a series of 104 operative cases, most of which have been done at the Children's Orthopædic Hospital in Seattle, it would appear that the operation has offered a cure in 13 cases, approximately 10 per cent. (duration of observation four years) and been of marked benefit in 50 per cent. The technique for the operation has not been perfected, nor is it yet known what cases will be benefited. Apparently cases in which there is marked cerebral atrophy are not particularly improved, at least no more so than by withdrawing the fluid intake. The most favourable cases are those in which there are demonstrable accumulations of cerebrospinal fluid but not particular enlargement of the ventricles.

R. G. G.

[80] **Late epilepsy** (Über Spätepilepsie).—E. KRAPF. *Arch. f. Psychiat.*, 1932, cxvii, 323.

THIS is a long and well-documented study of the epilepsy that appears late in life, with a full bibliography. It is based on the analysis of some 35 cases of socalled 'involution epilepsy.' In a narrow sense epilepsy as a symptom of senile change in the parenchyma itself is an altogether improbable occurrence, but the arteriosclerotic variety is common. Its relation to cardiac and vascular disorders, to vascular cerebral lesions, and to derangements of nutrition of the same derivation is discussed. Arterial hypertension is claimed to be a potent cause of the condition, and a direct one. Yet the author is unable to get away from the conception of an 'epileptic disposition' (whatever that may be) acting as a supplement to the vascular factor.

S. A. K. W.

[81] **The distribution of calcium between blood and fluid and the carbon dioxide content of the blood in epilepsy**.—S. KATZENELBOGEN. *Jour. Nerv. Ment. Dis.*, 1931, lxxiv, 636.

THIS somewhat inconclusive study from the Phipps Institute showed the following results:

The alkali reserve, determined in 31 dispensary epileptic patients, ranged between 54-74, extreme normal limits.

Blood calcium was measured in 86 ward patients and in 26 out-patients. It was found to be within normal limits (9-11 mgm. per cent.) in the out-patients, and 8·1-11 mgm. per cent. in the ward patients. In this last-named series of 86 cases, eight showed subnormal values, ranging between 8·1-8·9 mgm. per cent.

Calcium in the cerebrospinal fluid was found to be within 4·3-5·8 mgm. per cent. It was below normal in one case and above the normal in two out of 86 cases.

The alkali reserve figures and the blood calcium content of the author's epileptics, apparently, do not contribute to the concept of a basic relationship between epilepsy and tetany.

The fluid Ca/blood Ca ratios were within the limits 46-65. Ratios which may be considered as high normals were found in seven cases, and ratios distinctly above the normal in four cases.

No relation could be found between the calcium findings (in blood, fluid and ratios) and the mental state of the epileptic patients; nor have these findings, apparently, been influenced by the occurrence and frequency of convulsions, in so far as the latter took place within the two days before blood and spinal fluid were withdrawn.

The high fluid Ca/blood Ca ratios in 11 out of 86 cases and none with low ratios, suggest that there is in epilepsy a tendency to an increased permeability to calcium of the barrier between blood and fluid.

R. G. G.

[82] **Observations on some visceral conditions in general epilepsy and in convulsions of experimental origin.**—Samuel M. Weingrow. *Amer. Jour. Psychiat.*, 1932, xi, 737.

In animals after various surgical procedures upon the nervous system, after intravenous injections of glucose, tapwater, adrenalin, ammonium carbonate, luminal and potassium borotartrate, as well as after adrenalectomy and nephrectomy, there is an approximation of the minimal convulsive to the lethal doses of absinthe in experimentally produced convulsions as compared with control animals. When tapwater or ammonium carbonate is injected intravenously the ' convulsive factor ' is almost zero per cat and per pound as compared with 0·21 and 0·03 gm. respectively in the control animals. There seems a reduction in the number of the fits after adrenalectomy. Dilatation of the stomach is the most prominent symptom observed, that of the colon next, and contraction of the colon next. Some central and peripheral lesions of the nervous system show constant effects in dilatation or contraction of the parts of the digestive tube. These are not necessarily in

accordance with accepted neurophysiological principles. Emptying of the gall-bladder does not seem to be affected by the violent respiratory reactions. The occurrence of widespread and extensive petechial hæmorrhages in the lungs and congestion of other organs is a constant finding in all groups of experimentally produced convulsions.

C. S. R.

[83] **Experimental researches on subcortical epilepsy** (Recherches expérimentales sur l'épilepsie sous-corticale).—G. MARINESCO, O. SAGER, and A. KREINDLER. *Bull. soc. romaine neurol. et psychiat.*, 1932, xiii, 17.

IN these experiments an attempt was made to reproduce the clinical features of tonic epilepsy. Cats were chosen for the purpose, and choline was administered by intraventricular injection (1 mg. in 0·1 c.c. of Ringer's solution). Tonic attacks occurred, with trembling, and were accompanied by salivation, mydriasis, and incontinence. After the attacks the animals fell into a deep sleep.

In another cat, deprived previously of its cortex by two operations, and still living after eight months, analogous attacks occurred seemingly in a spontaneous fashion. It also, however, manifested symptoms like those of fugue, without fits, and affective attacks of rage.

The authors believe that their work tends to show that periventricular vegetative centres play an important part in the genesis of the epileptic fit, and that the tonic phase is of subcortical origin.

S. A. K. W.

[84] **The permeability of the hæmato-encephalic barrier in epilepsy, as determined by Walter's bromide test.**—S. KATZENELBOGEN. *Jour. Nerv. Ment. Dis.*, 1932, lxxv, 144.

THE distribution ratios of bromide between blood and cerebrospinal fluid in epilepsy was found to be within normal limits in 42 out of 87 cases (48·3 per cent.).

A distinct increased barrier permeability was observed in 12 out of 87 epileptic patients (13·8 per cent.).

A moderate rise above the normal permeability was found in 24 out of 87 cases (27·6 per cent.).

A decreased permeability was noted in nine out of 87 cases (10·3 per cent.). There was a marked decrease in three, and a moderate decrease in six cases.

As tested by Walter's method, the barrier permeability in epilepsy is likely to be found either normal or more or less increased in most cases.

There are relatively few chances to find a decreased barrier permeability to bromide in epilepsy.

The tendency toward a high permeability to bromide in epilepsy and the particular efficacy of the bromide treatment in this disease concur in contributing, apparently, to the concept that substances introduced into the general circulation affect the cerebrospinal system through the fluid.

The susceptibility of the barrier to a high permeability in epilepsy is also significant when compared with the frequent observation of an increased permeability in certain organic diseases of the cerebrospinal system and in conditions associated with meningeal congestion.

R. G. G.

[85] **Sodium chloride and water balance in epilepsy.**—F. PROESCHER and W. S. THOMAS. *Jour. Nerv. Ment. Dis.*, 1931, lxxiv, 577.

THE chlorine balance in adult epileptics shows a marked disturbance. Experimental evidence points to the existence of an abnormal tendency to retain sodium chloride. The capacity to store sodium chloride varies within wide limits and there seems to be no relationship between the number of convulsions or equivalents in the mental condition and the amount of sodium chloride stored. In some cases moderate or large doses of sodium chloride have the tendency to favour convulsive attacks or equivalents. According to experimental and pathological investigations the excess sodium chloride is probably stored up in the integument without consecutive water retention. The controls under the same experimental conditions showed no retention. From the data available at present the authors do not feel justified in giving an explanatory theory as to why the sodium chloride is stored up. The possibility of a central or endocrine disturbance is most likely because disturbance of the renal excretion cannot be demonstrated. If further investigations confirm the salt retention in all forms of the convulsive state, the rest may be of diagnostic value, especially in cases where no convulsions or equivalents are noted but where mental upsets of a doubtful nature exist.

The mechanism of the water balance in epilepsy is still obscure. From the data reported there seems to be no disturbance of the renal water output, while the extrarenal output varies in rather wide limits. A water retention could not be demonstrated definitely.

R. G. G.

86] **Basal metabolic rate in epilepsy.**—L. A. DAMON. *Arch. of Neurol. and Psychiat.*, 1932, xxviii, 120.

THE metabolic rates of 300 epileptic patients were tested and about half of the cases showed abnormal rates, the number of abnormal minus readings being about twice the abnormal plus readings.

R. M. S.

[87] **Clinical and experimental observations on the Babinski reversal, cardiovascular reactions, respiratory and pupillary changes during the convulsive and post-convulsive stages of general and experimental epilepsy.** —J. NOTKIN, HELEN C. COOMBS, and F. H. PIKE. *Amer. Jour. Psychiat.*, 1932, xi, 679.

IN the convulsive attacks in 69 epileptics the Babinski sign was positive in 65, i.e. 94·2 per cent., after cessation of the clonic movements. In 37 (56·9 per cent.) it became positive immediately after the last convulsive movement. In 28 instances (43·1 per cent.) the sign was positive two or three minutes after cessation of the convulsion. During this lapse there was no response at all, or occasionally even a flexor response. In 17 observations (26·1 per cent.) the sign could be elicited on one side only; in the other 48 cases it was elicited on both sides. Its duration is from half a minute to 25 minutes and appears to increase with the age of the patient. Its mechanism is still in the realm of speculation. The writers suggest a transitory derangement of the pyramidal system with a consequent appearance of pyramidal tract signs of a fleeting character.

Immediately after the convulsion there is a pronounced tachycardia with a gradual decrease in pulse-rate until the recovery of the interparoxysmal rhythm in 5 to 30 minutes. The systolic pressure rises at once after the seizure in about half the cases, and in the other half it is decreased. The diastolic pressure remains normal in the cases where the systolic pressure is increased after the fit, but decreases markedly with a decrease in systolic pressure. Experimental results show a rise of blood-pressure and generally of heart rate in intact, unanæsthetized animals after intravenous injection of absinthe or camphor monobromide in sufficient doses to elicit a well-marked convulsion. Neither the increase of blood-pressure nor of heart rate is invariable. The fact that no increase of blood-pressure occurs in animals experimentally when the skeletal muscles are eliminated by the intravenous injection of curare, or when the spinal cord is transected high in the thoracic region, shows that such rises of blood-pressure as do occur in other animals are due to the action of the striated musculature during a clonic convulsion. A similar rise of blood-pressure does not occur when the convulsant is injected within a few hours after bilateral ablation of the cortical motor areas; the convulsions under these conditions are tonic. Nor does the rise of blood-pressure occur in control animals when the dose of the convulsant agent is such that a tonic and not a clonic convulsion appears. The wide variability of cardiovascular changes in the human subject during epilepsy indicates a lack of any constant or specific primary action upon the cardiovascular mechanism.

Under experimental conditions, there is usually an increase in the respiratory rate at subconvulsive doses of the convulsant. At the convulsive

level the record of respiration is obscured by clonic movements. After sublethal doses the subsidence of the convulsive movements is followed by a period of rapid respiratory movements. When the dosage reaches the value at which tonic phenomena are elicited, the respiration is slowed, and may even be intermitted during the period of greatest tonic activity. The pupils are dilated and rigid to light during both phases of the fit. At the early phase of relaxation they may be unequal and react sluggishly to light. In the second or third minute of relaxation they may become pin-point, giving paradoxical light reactions. The reaction of the pupil to light returns before the Babinski sign disappears, and before the pulse-rate and blood-pressure return to normal. Under experimental conditions, there is also dilation and rigidity of the pupils to light, but the corneal reflex may be present at times while the pupil is still rigid. When one cervical sympathetic nerve is divided some hours before the experiment, the pupil on the same side contracts under absinthe, while the other dilates. The usual response of the pupil to convulsants is decreased when the cortical motor areas are removed a few hours before.

C. S. R.

SENSORIMOTOR NEUROLOGY.

[88] **A study of the eye findings in a group of epileptics, with reference to the mechanical theory of epilepsy.** —Harry Costeff. *Amer. Jour. Psychiat.*, 1932, xi, 747.

The fundi of 114 epileptics were studied with the object of ascertaining the presence or absence of increased intracranial pressure. Of this number 58 showed a distinct pathology, while nine could not be examined. The most frequent finding was a venous engorgement of the eye fundus. This group constituted 55·1 per cent. of the total. Of these 32 cases of passive hyperæmia 18 persisted, while 14 occasionally cleared up during the interparoxysmal period. Temple Fay's mechanical theory of epilepsy is reviewed and discussed. The work of Kalt, Claude, and others on the relation between intracranial pressure and the pressure in the retinal vessels, is also referred to. The conclusion is reached that the relatively frequent incidence of passive hyperæmia in the fundus of epileptics would tend to prove the existence of intracranial hypertension in at least a considerable number of cases.

C. S. R.

[89] **Multiple tumours of the nervous system.**—H. L. Stewart. *Arch. of Neurol. and Psychiat.*, 1932, xxviii, 132.

A case of multiple tumours of the nervous system is reported in which each differed from the other in the type and arrangement of the cells, number of mitotic figures, construction and number of blood-vessels, amount of necrosis and kind and quality of fibres manufactured. Evidence is presented that all these tumours originated from the same embryological tissue.

R. M. S.

[90] **Hemiballismus** (Hemibalismo, a proposito de tres casos).—E. Moniz. *Lisboa Medica*, 1931, viii, 481.

The conclusions of this paper are that hemiballismus may occur without hemiplegia, and is usually caused by vascular lesions of the regio subthalamica. It is subject to the laws governing the symptomatology of vascular lesions and may therefore much improve clinically, or even be cured. It may also be induced by tumours, directly or indirectly. Included in the area where lesions are prone to produce it are the corpus Luysii and locus niger. To understand its pathogenesis the connexions of the motor system with thalamus, corpus striatum, nucleus dentatus, etc., have to be borne in mind. If the cortical centres and their system are involved hyperkinesis will not occur.

Satisfactory results are claimed for the use of genoscopolamine in the condition.

S. A. K. W.

[91] **Convulsive manifestations in Huntington's chorea.**—J. Notkin. *Jour. Nerv. Ment. Dis.*, 1931, lxxiv, 149.

Cases of Huntington's chorea with convulsive manifestations reported in the literature are reviewed and one personal observation is described in detail. The presence of various pathological lesions (in the reviewed cases) in addition to the usual findings in corpus striatum and cortex, make it impossible to ascribe the convulsive attacks to a definitely localized lesion. On the other hand the organic process in the disease, which is probably active for years before the appearance of motor and mental manifestations, may prepare the terrain for the convulsive mechanism. There seems to be some relationship between Huntington's chorea and convulsive states since they frequently appear in collaterals.

R. G. G.

[92] **Pelizæus-Merzbacher disease (familial centrolobar sclerosis).**—Y. SCHEFTEL. *Jour. Nerv. Ment. Dis.*, 1931, lxxiv, 175.

Two cases are described and the literature reviewed. The differential diagnosis between this and other forms of cerebral sclerosis—diffuse gliosis, multiple sclerosis, Tay-Sach's disease and Schilder's disease—is discussed. The two cases described are included under the heading of Pelizæus-Merzbacher disease on account of the following features: familial incidence—it was noteworthy that females greatly predominated in the family— and its outstanding symptoms, viz., speech disturbance, incoordination of movements or ataxia, athetosis, intention tremor, clonus, spasticity, intellectual retardation and psychic peculiarities. Abnormal reflexes such as Babinski and ankle clonus were elicited inconstantly, while the abdominals varied also. The same is true of the choreiform movements and of the nystagmus. There were no sphincter or sensory disturbances. The onset was in infancy in the older and at about six years in the younger boy. In both cases remissions have occurred. The clinical findings point to subcortical extrapyramidal lesions of cerebrum and cerebellum.

R. G. G.

[93] **Congenital hypoplasia of the olivopontocerebellar tracts.**—H. M. ZIMMERMAN and K. H. FINLEY. *Arch. of Neurol. and Psychiat.*, 1932, xxvii, 1402.

THE history is presented of a female infant, aged 13 months, who was delivered in the eighth month of pregnancy by cæsarean section because of complicating nephritis in the mother, aged 42. At birth, a spina bifida that was present was repaired, and a hydrocephalus was noted, which increased in severity until the child died. Death occurred with the sudden onset of dyspnœa followed by a convulsion of the right side. Throughout life the child had a spastic quadriplegia.

Necropsy revealed a marked communicating hydrocephalus and a fresh hæmorrhage over the left cerebellar hemisphere. The latter was atrophic, which was true as well for the left dentate nucleus and the right pyramidal tract. The entire vermis was malformed, and the central medullary nuclei of the cerebellum were absent, being replaced by several cortical cerebellar heterotopias. In association with the cerebellar lesions was an atrophy of the left restiform body, both dorsal accessary olives, the whole right inferior olive, the ventral limbs of both inferior olives, the median accessory olives, the right pontine nuclei and the left middle and superior cerebellar peduncles. There was an atrophy of the right Deiters' and the right dorsal glossopharyngeal and the left ambiguus nuclei.

It is concluded that the atrophy of the right inferior olive was dependent on that of the left cerebellar hemisphere; that the dorsal accessory olives are connected with the dorsal part of the vermis, and that the median accessory olives and the ventral limbs of the inferior olives are connected with the ventral part of the vermis.

Related to the atrophy of the left cerebellar hemisphere was a hypoplasia of the right pontine nuclei and the left middle cerebellar peduncle.

Because the left dentate nucleus was atrophic the left superior cerebellar peduncle, as well as the right red nucleus, was smaller than the corresponding structures of the opposite side. The atrophy of the right nucleus of Deiters and of the right dorsal glossopharyngeal and the left ambiguus nuclei could not be satisfactorily explained or related to the other observations.

R. M. S.

[94] **Brain involvement in head injury.**—W. Ritchie Russell. *Edin. Med. Jour.*, 1932, xxxix, 25.

Dr. Ritchie Russell here communicates the salient features of his study of 200 cases of head injury admitted to the Royal Infirmary in Edinburgh. All were examined from a neurological point of view. His clinical descriptions are useful, and his tables informative. Among other points of interest we may note that from 67 examinations of the spinal fluid he obtained figures indicating a pressure below 100 mm. of water in 7, between 100 and 200 in 18, between 200 and 300 in 40, and over 300 in 14. Blood in the spinal fluid occurred as follows: a count of less than 1,000 reds per c.mm. in 35; from 1,000 to 100,000, in 38; over 100,000, in 9. Dr. Russell attributes the loss of consciousness to commotio of nerve-elements and advances cogent arguments against the view that it is due to cerebral anæmia caused by sudden flattening of the skull. Treatment is also discussed at some length.

J. V.

[95] **Alternating oculomotor paralysis in traumatic middle meningeal hæmorrhage.**—E. S. Gurdjian. *Arch. of Neurol. and Psychiat.*, 1932, xxviii, 26.

The author quotes four cases to show that acute traumatic middle meningeal hæmorrhage may give rise to typical alternating oculomotor paralysis by virtue of the fact that the extension of the dissecting clot into the base of the middle fossa causes an oculomotor palsy, and its pressure against the convexity of the hemisphere is productive of contralateral paralysis of the body. From the surgical standpoint it is important to localize such a lesion accurately, and it is suggested that when the diagnosis of middle meningeal hæmorrhage is made, an osteoplastic flap should be the operation of choice.

It is always wise to drill a little hole or two in the bone to confirm the diagnosis. These openings should be made in close proximity to the ear, so that a possible clot that has not extended further than the base of the middle fossa may not be missed.

R. M. S.

[96] **The diagnosis of cerebral tumour** (Die Diagnostik des Tumor cerebri). —B. Brouwer. *Psychiat. en Neurol. Bladen*, 1932, xxxiv, 1.

Professor Brouwer has collected and analyzed the results of his experience with cerebral tumours from the period 1923-1931 inclusive. The number of cases amounted to 336. Interesting summaries are given for which the original paper should be consulted, in reference to site and variety, malignancy, special features of different types and places, and so forth. Professor Brouwer gives a long list of other morbid states with which brain tumour may be confused—17 in all; each of these is annotated.

S. A. K. W.

[97] **Osteitis fibrosa cystica of the skull.**—H. D. Palmer, R. Harrow and L. A. Schwartz. *Arch. Neurol. and Psychiat.*, 1932, xxvii, 45.

A case of osteitis fibrosa cystica localized in the occipital bone and accompanied by visual hallucinations and left homonymous scotoma is described by these writers. At two years of age a fracture of the occipital bone occurred. By the age of twelve years depression, dream states, periods of amnesia and auditory and visual hallucinations were present. Right occipital craniotomy and removal of the inner wall of the cyst was followed by the complete disappearance of the hallucinations and mental symptoms.

G. de M. R.

[98] **Ventriculography and encephalography.**—F. C. Grant. *Arch. of Neurol. and Psychiat.*, 1932, xxvii, 1310.

Reviewing 325 encephalograms and 160 ventriculograms, the author concludes that the replacement of the subarachnoid fluid by air is of much value both in diagnosis and in prognosis in intracranial disease; in certain conditions the therapeutic results justify its use. In patients with increased intracranial pressure suspected of having a tumour of the brain the air should be introduced by direct bilateral ventricular tap and not by lumbar puncture. Ventriculography gives more accurate information as to the position of a mass lesion with less distress and greater safety to the patient than does encephalography. But in patients without increased tension, for a differential diagnosis between atrophy, sclerosis and tumour, or between traumatic neurosis and true posttraumatic lesions, for prognosis in epilepsy, birth

trauma or degenerative disease, or for therapy in posttraumatic headache, an outline of the lateral ventricles and subarachnoid channels should always be obtained by encephalography.

R. M. S.

[99] **Cerebral changes in pneumococcus septicæmia.**—H. M. ZIMMERMAN and H. YANNET. *Jour. Nerv. Ment. Dis.*, 1932, lxxv, 386.

A CASE is described of pneumococcal septicæmia with the following cerebral symptoms. The child had frequent convulsions which were difficult to control with the ordinary sedative drugs. Spasticity of the right arm and leg became more marked, and on the seventh day of illness dysphagia developed. The child died on the ninth day with a clinical diagnosis of lobar pneumonia of the upper lobe of the right lung, septicæmia, and encephalitis.

Post-mortem examination revealed a bilateral diffuse pneumonia and fibrinous pleurisy, and no gross changes in the brain. Histologically, the cerebral lesions consisted of a degeneration of the nerve-cells of the cortex and central grey matter, sometimes focal and sometimes diffuse in distribution. The degeneration was in the nature of the ' acute swelling ' of Nissl. The smaller blood-vessels in the cortex and central grey regions had swollen endothelium and their walls had a peculiar hyaline translucency. These changes appeared to have a positive association with the nerve-cell changes. Nowhere in the brain was there a leucocytic reaction. Active protoplasmic glia proliferation was found in the zones of ganglion-cell destruction and to a lesser degree in the white matter. Demyelination, perivascular and otherwise, was present in the cortical grey matter and more conspicuously in the centrum semiovale. In the zones of myelin destruction was scattered much fat, some free and some phagocytosed.

R. G. G.

[100] **Cerebral arteriovenous aneurysms with report of a case.**—F. P. MOERSCH and J. W. KERNOHAN. *Jour. Nerv. Ment. Dis.*, 1931, lxxiv, 137.

THE general features of the condition are discussed and a somewhat unusual case fully quoted. This is a comparatively rare anomaly usually of congenital origin and characterized by a fairly constant chain of clinical symptoms, which usually extend over a long period and show a marked tendency to fluctuation. The condition is most frequently encountered in young adults. A bruit is a fairly constant but not a diagnostic sign. An X-ray of the head will usually disclose calcium deposits in the wall of the aneurysm. A study of the blood from the jugular and median veins may help to establish the presence of an arteriovenous fistula.

R. G. G.

[101] **Subacute necrotic myelitis with lesions of the optic nerve** (Un cas de myélite nécrotique subaigue glio-angeio-hypertrophique, avec lesions du nerf optique).—I. Minea. *Bull. soc. romaine neurol. et psychiat.*, 1932, xiii, 27.

This report concerns the case of a man of 23, with total paraplegia developing in the course of some three months. Five years previously he had lost the sight of the right eye in the course of a few days, while that of the left became dim; an internal strabismus of the right eye followed. On examination there was complete loss of sensibility downwards from the level of the tenth thoracic roots. Lumbar puncture gave a slightly xanthochromic fluid, with 58 lymphocytes and a negative Wassermann test. The patient died ten days after admission.

Lesions of a peculiar kind distinguished the case. The vessels of the spinal meninges and cord showed an extraordinary hypertrophy of the adventitia, with thickening of middle and inner coats also; many were occluded. Irregular foci with pronounced reaction of the macroglia were seen—the condition might be described as an angiohypertrophic gliosis. The resemblance of the case to those reported by Foix and by Lhermitte was close.

S. A. K. W.

[102] **Neurological aspects of primary anæmia.**—R. S. Ahrens. *Arch. of Neurol. and Psychiat.*, 1932, xxviii, 92.

In a study of 189 cases of proved primary anæmia it was found that 97·77 per cent. showed evidence of combined degeneration of the spinal cord as given by posterior and lateral column signs. The author concludes that if carefully examined all cases would probably show cord changes at some time during the course of the disease. With regard to liver therapy, the impression was gained that this has little or no effect on the pathology of the central nervous system.

R. M. S.

[103] **Brown-Séquard paralysis.**—G. B. Hassin, F. J. Kendrick, and E. J. Connelly. *Jour. Nerv. Ment. Dis.*, 1930, lxxii, 245.

It is unusual to find the Brown-Séquard syndrome following inflammatory lesions. Two cases of such sequence are here described, one of poliomyelitis and one of syphilitic myelitis.

R. G. G.

[104] **Sciatica: clinical forms and treatment of rheumatismal sciatica.**—H. ROGER. *Jour. Nerv. Ment. Dis.*, 1931, lxxiv, 1.

THE author enumerates the varied causes of pain referred to the back of the leg. He points out that four syndromes may be recognized: (1) a sensory syndrome of subjective pain and pain elicited by pressing on various points and on stretching the nerve; (2) a motor syndrome including abnormal postures taken by the patient, muscular tonicity, muscular atrophy, disturbances of electrical reaction, motor deficiency and reflex disturbances; (3) a sympathetic syndrome with vasomotor and thermal disturbances, abnormalities of sweating and trophic and pilomotor anomalies; (4) a humoral syndrome involving in some cases a slight meningeal reaction in the cerebrospinal fluid. He describes five clinical types, the last two not being actually associated with lesions of the nerve.

(1) *Lumbar sciatica:* the lumbar roots are involved, the pain is 'high,' and stretching of the nerve makes little difference. This is easily confused with various other conditions, especially intrathecal.

(2) *Sacral sciatica:* due to involvement of the sacral plexus, a common form which has to be differentiated from pain referred from an arthritis of the hip or sacroiliac joint.

(3) *Truncal sciatica:* true sciatic neuritis or perineuritis, with the typical classical signs.

(4) *Myalgic sciatica:* really a fibrositis or myositis.

(5) *Cellular sciatica:* due to the infiltration of cellular products and the formation of nodules.

R. G. G.

[105] **Polyneuritis after use of an abortifacient (triorthocresyl phosphate poisoning)** (Polyneuritis nach Gebrauch eines Abortivums: Triortho-kresyl-phosphat-Vergiftung).—J. W. G. ter BRAAK and R. CARILLO. *Deuts. Zeits. f. Nervenheilk.*, 1932, cxxv, 86.

TOWARDS the end of 1930 and beginning of 1931 some 37 cases of an unusual form of polyneuritis occurred in Holland. Clinically, they were distinguished by the following features: the almost purely motor character of the symptoms; a symmetrical predilection for the muscles of distal segments of arms and legs; loss of ankle jerks with preservation or even exaggeration of knee-jerks. The cause was ascertained to be the use of 'Apiol' as an abortifacient—a preparation consisting essentially of triorthocresyl phosphate. The polyneuritis thus comes into line with that known to be produced by creosote phosphate, and with socalled 'ginger paralysis.' Experiments on fowls were conducted and the histological nature of the condition fully established; it constitutes a periaxial neuritis. Myelin sheaths are first affected, and axons secondarily.

S. A. K. W.

[106] **Cheiralgia paræsthetica** (Cheiralgia paræsthetica; isolierte Neuritis des Ramus superficialis nervi radialis).—R. WARTENBERG. *Zeits. f. d. g. Neurol. u. Psychiat.*, 1932, cxli, 145.

THE condition for which the name 'cheiralgia paræsthetica' is suggested (a term, surely, rather more comprehensive than is required) is a sensory mononeuritis of the end twig of the nervus radialis that supplies the ulnar side of the thumb; the radial side of the hand and of the forefinger may also be involved distally. Subjective and objective sensory disturbances are present and may be pronounced, but deep sensibility is unimpaired. When the nerve concerned is put on the stretch pain is always elicited. The cause of the neuritis seems to be rather variable. A number of analogous cases are cited from the literature.

S. A. K. W.

[107] **Amyoplasia congenita.**—WILFRED SHELDON. *Arch. of Dis. in Childhood*, 1932, vii, 117.

DR. SHELDON describes the case of a child with congenitally rigid arms and legs, associated with aplasia of certain muscle groups. From the literature he has collected and analyzed some 44 cases, and adds five more. In his view the initial lesion consists of this developmental aplasia, with secondary changes in joint structure. He prefers the name quoted above to the synonyms of 'arthrogryposis multiplex congenita' and 'multiple congenital articular rigidity.'

J. V.

[108] **Dermatomyositis** (Contribution à l'étude de la dermato-myosite; examen anatomo-clinique d'un cas).—G. MARINESCO, S. DRAGANESCO and E. FACON. *Ann. de médecine*, 1931, xxx, 145.

THE case described is that of a youth of 18, a chauffeur, who was admitted to hospital complaining of weakness of limbs, pains in arms and thighs, and difficulty in swallowing. The symptoms were of four months' duration. On examination, œdema and erythema were observed over the face and elsewhere; a moderate rise of temperature was noted. Biopsy of a fragment from the biceps showed the characteristic lesions of dermatomyositis. About a month after admission the patient died from asphyxia, secondary to an inhalation pneumonia.

The nervous system proved to be in a state of hyperæmic congestion. Nearly all the skeletal muscles were reduced in volume, or atrophic, firm, pale, and infiltrated with connective tissue. Microscopically, the skin of the face was found to be the seat of lymphocytic collections round small vessels, with numbers of clasmatocytes round sweat-ducts and elsewhere. The capillaries were all dilated; small hæmorrhagic effusions were also seen.

A full description is given of the inflammatory changes in the muscles. In the peripheral nerves small plaques of demyelination were discovered, with hyperplasia of the cells of the sheath of Schwann.

The observers exclude erysipelas, trichinosis, sclerodermia, and polyneuritis, also the rare myopathic poikilodermia of Petges. The nature of the infection remains obscure.

S. A. K. W.

PROGNOSIS AND TREATMENT.

[109] **Anionic bismuth therapy in neurosyphilis.**—H. G. MEHRTENS and P. S. POUPPIRT. *Amer. Jour. Syphilis*, 1932, xvi, 373.

THE work of L. Stern and her associates has shown that anions pass more readily than kations from the blood to the brain and cerebrospinal fluid. An attempt was therefore made to assess the passage of bismuth when used in an electronegative form in therapeutic doses.

In previous experimental work very large, often fatal, doses were given, which might well have passed the cerebral barrier by poisoning it. In the present series of experiments sodium and ammonium iodobismuthite, when given to guinea pigs intramuscularly or intravenously in doses of therapeutic range, was found in the brain in concentrations which averaged 0·05 mgm. per 100 grammes. Neither intravenous injections nor the simultaneous administration of iodide increased the concentration of bismuth in the brain. Sodium bismuth tartrate passed into the brain in approximately half this concentration, and products containing electropositive bismuth appeared to pass still less readily. The brain contained at least as high a concentration as the blood with electronegative preparations, but a much lower concentration than the kidneys.

An investigation was then undertaken to determine whether anionic bismuth passed into the cerebrospinal fluid of patients more readily than the kationic salts. Previous investigators had found that apart from meningitis or some other complicating factor, bismuth did not pass into the cerebrospinal fluid, and the present authors confirmed this for the kationic salts in 73 per cent. of their patients. On the other hand, of patients undergoing treatment with sodium iodobismuthite, 83 per cent. were shown to have bismuth in the cerebrospinal fluid. When the total dosage of iodobismuthite reached 0·72 grammes, bismuth was almost always found in the fluid. Bismuth was also found in the brains of six cases receiving iodobismitol and of three who had received bismuth sodium tartrate and other forms of bismuth.

The clinical results obtained in the various forms of neurosyphilis by the use of anionic bismuth preparations were encouraging.

J. G. G.

[110] **Case of pneumococcus (type III) meningitis treated with potassium permanganate; recovery.**—M. H. WEINBERG. *Jour. Nerv. Ment. Dis.*, 1931, lxxiv, 38.

THE case is described of a patient who was said to be moribund when treatment by an aqueous solution of potassium permanganate given by the rectum according to the method of Nott was commenced. Recovery resulted. The author believes that the manganese ion acts as a detoxicant of the pneumococcal virus and that this allows the natural immunizing agents to exert their influence.

R. G. G.

[111] **Atlanto-occipital evacuative trepanation in contusions of the brain.**—F. ODY. *Arch. of Neurol. and Psychiat.*, 1932, xxviii, 112.

ODY considers that in traumatic lesions of the brain atlanto-occipital trepanation with liberation of the posterior arch of the atlas is the operation of choice. The advantages are:—

(1) The operation renders aid not only at the site of the primary lesions, but where it is most essential to give relief, that is, to the medulla, whose centres are imperilled by peribulbar clots.

(2) It allows the drainage of blood extravasated in the subarachnoid spaces even when the primary lesions cannot be exactly localized.

(3) This method of drainage is more effective than the classic temporal procedure. It is made at the most dependent point and from a large lake, whereas the latter is from a region where no physiological accumulation of fluid exists, and where the blood can exude only by infiltration.

(4) In atlanto-occipital trepanation, postoperative hernia of the brain does not occur as in temporal drainage. If a foraminal herniation of the cerebellum should result from increased intracranial pressure, resection of the posterior arch of the atlas would free the strangulated cerebellar tonsils after evacuation of the fluid.

(5) The integrity of the skull is preserved.

(6) This operation is simple, and necessitates no special instruments.

R. M. S.

[112] **Experiences in the treatment of multiple sclerosis with quinine hydrochloride.**—R. M. BRICKNER. *Arch. of Neurol. and Psychiat.*, 1932, xxviii, 125.

EXPERIENCES with the use of quinine hydrochloride in the treatment of sixteen patients with multiple sclerosis over a period of one year are given. The theory of quinine therapy rests on the hypothesis, suggested but not yet

proved, that the lesions are caused by abnormal lipolytic activity of the blood, and that this activity may be due to the presence of an abnormal lipase. The effects of the therapy were studied symptom by symptom. Forty symptoms, most of them of short duration, showed marked improvement. Thirty-three symptoms did not improve; most of the latter were old. All of the improvements but one were maintained at a fairly constant level, and only three of the unimproved symptoms regressed. Specificity in the effect of quinine is suggested by the relative fixity of the interval between the initiation of treatment and the beginning of improvement; the grouping, by duration, of symptoms which have and have not improved; the fact that the most recent symptoms have improved not only the most promptly, but also the most completely.

R. M. S.

[113] **Poliomyelitis.**—I. J. Sands. *Jour. Nerv. Ment. Dis.*, 1932, lxxv, 616.

A general survey of the clinical and pathological picture of this disease. The following conclusions in reference to treatment may be noted.

Treatment should be directed towards neutralization of the virus by intravenous and intramuscular administration of serum from convalescent patients as soon as the diagnosis is made; reduction of cerebral œdema by administration of hypertonic glucose solutions intravenously, and by spinal drainage; combating difficulties in respiration and in deglutition; and protecting the paralyzed muscles by proper mechanical support.

The treatment of the paralyses should be directed by properly trained physicians and by competent nurses trained in orthopædic and physiotherapeutic principles.

Prophylactic measures during an epidemic may be advantageously utilized by segregating the child, and by the administration of immune serum or whole blood intramuscularly. The latter procedure may be repeated every three or four weeks.

R. G. G.

[114] **Surgical intervention in four cases of compression-myelitis caused by osseous deposits in the arachnoidea of the spinal cord (arachnoiditis ossificans).**—L. Puusepp. *Jour. Nerv. Ment. Dis.*, 1931, lxxiii, 1.

The author describes four cases dealt with by surgical means. He points out that the root pains which are not always present and other symptoms referable to the spinal cord are due to pressure either from bony plates or from partially ossified connective tissue or to collections of fluid under the plates. This compression may be successfully removed, as in the four cases referred to, by surgical means. The lipiodol method is useful in the diagnosis of the condition.

R. G. G.

[115] **A consideration of sacral epidural injections for relief of sciatic pain.**—F. G. Lindemulder. *Jour. Nerv. Ment. Dis.*, 1931, lxxiv, 265.

Forty-six patients suffering from sciatic pain received epidural injections through the sacral hiatus of 1 per cent. procaine hydrochloride in Ringer's solution; of these 40 were relieved. Foci of infection when present were removed as a probable cause of the sciatic pain. In four cases in which the achillis jerk was absent, the reflex returned after treatment.

R. G. G.

Psychopathology.

PSYCHOLOGY.

[116] **Cardio-respiratory variations in personality studies.**—J. A. Larson and G. W. Haney. *Amer. Jour. of Psychiat.*, 1932, xi, 1035.

Primarily there is a discussion of psychogalvanic technique results on general and pathological problems, followed by a review of the literature on the relation of basal metabolism to personality type or deviation. This paper is a study of the relative rôle which physical criteria, as obtained by the use of polygraph methods, may play in the psychiatric examination of children who present behaviour difficulties. The data presented are based upon the examination of 235 children, of whom 113 were referred by psychiatrists because of problems revolving about deception. It was found that the polygraph technique was of material aid in the detection of deception, and was also useful in securing rapport in children who were resistant to the usual approach. Such physical criteria were of value in investigating the deception of the psychoneurotic type of individual who may be attempting to convince himself that he suffers from certain ailments or that certain events have not happened. It was also valuable in the study of alleged hallucinatory experiences. The technique might prove just as effective in many cases as long drawn out psychoanalysis. After a study of 2,000 records, no definite reaction pattern was found which was finally diagnostic. It would seem that some records were more irregular in cases of psychopathology or actual cardio-respiratory pathology. There was some similarity between records of exhaustive states, chronic illness, catatonia, and cases of hypothyroid function, but there were no clear cut lines of identification. The study of respiratory and cardiac records together with the utilization of drugs and analytical methods offers a possible means of further study of the schizophrenic processes. Certain trends appear in these cardio-pneumo-psychograms which might be of service in personality analysis and classification. Such psychograms seem to afford a simple and often the most satisfactory method for studying complex situations and problems involving personality.

C. S. R.

[117] **Inconstancy of the formal structure of the personality.**—F. WERTHAM and M. BLEULER, *Arch. of Neurol. and Psychiat.*, 1932, xxviii, 52.

RORSCHACH'S ink-blot test was studied on normal subjects, both before and after the administration of mescaline. It was found that the test under mescaline corresponded closely to the personality of the subject as actually known. The test obtained in normal circumstances, while agreeing in some points, gave an opposed picture of all important qualities. The authors therefore suggested that their observations indicated that the form of a personality is not constant and that the usual psychological 'type' of a person does not necessarily describe completely the structure of his personality.

G. de M. R.

[118] **The formation of a simple habit in guinea-pigs.**—G. C. GRINDLEY. *Brit. Jour. Psychol.*, 1932, xxiii, 127.

AN attempt is here made to study 'trial-and-error learning,' when the response to be learnt is a relatively simple movement, and the stimulus for that response is also relatively simple. Guinea-pigs, placed in a stand in a sound-proof room, were taught to turn their heads to one side in response to the sound of a buzzer, food being given as a reward when they made this movement. It was found that the average time of response became progressively shorter in successive trials. The habit could be 'reversed' by rewarding the animal for turning its head, on the sound of the buzzer, to the other side; or 'extinguished' by ceasing to reward the animal.

The argument is that it is difficult to explain these results on Pavlov's theory of conditioned reflexes; and that it seems probable that some other principle, such as that of Thorndike's 'law of effect' or Troland's principle of 'retroflex action,' must be added to the principle of conditioning in order to explain the simpler forms of learning.

C. S. R.

PSYCHOSES.

[119] **Tactual perception in alcoholism.**—WALTER BROMBERG. *Arch. of Neurol. and Psychiat.*, 1932, xxviii, 37.

STUDIES of the after-effects of tactual sensations made on patients with alcoholic and schizophrenic psychoses showed that the tendencies toward change in the after-effects found in normal persons were present in the patients. In persons suffering from alcoholism the after-effects change in such a manner as to indicate that there are organic changes in the perceptive field. These organic changes may be in the nature of sensory or vestibular

influences. In alcoholism, changed sensory perception is basic for the existence of hallucinations. In patients with schizophrenia, there are no changes in the perceptive field; hallucinations among these patients are related to the special thought-processes of schizophrenia. It is still an open question in what way these special thought-processes influence secondarily the perceptual field.

R. M. S.

[120] **A study of psychogenic factors in thyrotoxicosis.**—H. E. Kiene and H. H. Dixon. *Jour. Nerv. Ment. Dis.*, 1931, lxxiv, 483.

The authors are of opinion that thyroid disorders occur in individuals of a personality which reacts to excitement and psychogenic trauma with the development of excessive and prolonged tension.

This tension is a physiological response of the organism which with constant overstimulation may produce permanent changes in the thyroid, and these changes render the individual more susceptible to subsequent physical and emotional trauma. The physiological response is of the type which, when constantly stimulated by psychogenic trauma, results in temporary and permanent changes in the thyroid gland and associated mechanisms, giving the clinical syndrome described as hyperthyroidism. The susceptibility of the thyroid gland to change is much increased by biochemical alterations such as occur when the iodine available is insufficient for the normal metabolic requirements of the gland.

Damage to the thyroid gland by infective and toxic processes contributes to the ease of permanent change. The fully developed syndrome represents a vicious circle which can be interrupted by thyroidectomy, but early cases offer therapeutic possibilities and relief may be gained by altering the factors to which the patient must react. In postoperative cases recurrences would probably not result if the tension could be relieved. Therefore, owing to the presence of underlying psychogenic factors of causal significance in hyperthyroid cases, they recommend complete psychiatric studies.

R. G. G.

[121] **Thyroid psychosis.**—S. D. Ingham and J. M. Nielsen. *Jour. Nerv. Ment. Dis.*, 1931, lxxiv, 271.

Although it has been claimed that psychoses in persons suffering from disturbances of thyroid function do not differ from those which occur in cases without thyroid involvement, the authors describe four in which ordinary treatment had no effect whereas treatment of the thyroid abnormality immediately effected a cure. They plead that toxæmia should never be overlooked in any case of psychosis.

R. G. G.

[122] **Paranoid reaction occurring in women of middle age.**—G. H. STEVENSON and S. R. MONTGOMERY. *Amer. Jour. Psychiat.*, 1932, xi, 911.

THERE appears to be a clinical sub-entity in the psychogenic or 'reaction-to-environment' group of psychoses, in which the chief symptoms are persecutory delusions, with or without hallucinations, and without obvious personality or intellectual changes, occurring in highly moral women, and becoming apparent during the fifth and sixth decades after a long prodromal period. The change from normal to psychopathic is so gradual that relatives have found difficulty in fixing the date of onset and persons who have but slight contact with the patient are not aware that the individual is suffering from a psychosis. The writers think that there is a specific etiological factor and that this is an overt or imagined sinful act on the part of the patient, which is not sinful to her, however, as she either projects the blame on others or denies that it would have been consciously desired by her. The situation is thus not dealt with adequately and honestly, and a state of emotional unrest is produced. As a quietus to conscience a persecutory delusional trend is developed slowly and progressively until a well-marked psychosis results.

C. S. R.

[123] **Principles of Gestalt in copied form in mentally defective and schizophrenic persons.**—L. BENDER. *Arch. of Neurol. and Psychiat.*, 1932, xxvii, 661.

MENTALLY defective persons from mental ages of between one and two years were studied and compared with cases of schizophrenia. The author found tendencies for Gestalt at the three-year level. Above three years there is a tendency to accentuate the horizontal base lines, to control perseveration and to produce wavy lines instead of broken ones for the representation of straight lines, and some effort to cross lines. At the five-year level, there is a tendency to reduce the primitive loops to points, but this tendency is reversible even at the superior adult level, and there is a tendency to make straighter lines and better recognized Gestalten. At the seven-year level, there is a swinging back and forth between loops and dots, a capacity for many vertical and geometric Gestalten determined on the principle of internal organization and for crossed forms. At the eight-year level, most Gestalten forms are satisfactory; slanting forms are recognized, but their relations to the whole figure are not always possible. Pairing determined by slight differences is not possible. The major difficulty at this level seems to be in connexion with the relations of parts to wholes. These things are satisfactorily accomplished above the ten-year level, though even then there is a certain amount of variation in individual cases. At all levels all of the original principles are in evidence and tendencies to revert back to

them are always present. Thus, there is always the tendency for dots to become loops, for straight lines to curve a little, for parallel lines to radiate toward or away from each other. In the schizophrenic cases there were found dissociations due to reversions back to some of the primitive principles such as perseverations, carrying over one form into the next, changing dots to loops, fragmentations, representing dotted lines as wavy lines, micropsia and accentuation of the horizontal plane, and to avoid crossed and angulated forms, as well as failure to integrate the whole field or properly relate some one or more parts to the whole.

G. de M. R.

[124] **Reflex response latencies in cyclothymic and schizophrenic cases.**—J. M. Dorsey, A. A. Mich, and L. E. Travis. *Arch. of Neurol. and Psychiat.*, 1932, xxvii, 687.

The patellar tendon reflex was studied by the authors, and it was found that the ratio of standing height divided by reflex time was a suitable unit of measurement.

Under this ratio 18 manic cases showed a mean ratio of 7·99 ± ·05, eight depressed cases showed a mean ratio of 8·52 ± ·06, 122 normal students showed a mean ratio of 8·76 ± ·02, and five cases of catatonic stupor gave a figure of 13·67 ± 2·40.

G. de M. R.

[125] **The acid-base equilibrium of the blood in psychotic patients.**—K. E. Appel, C. B. Farr and P. J. Hodes. *Jour. Nerv. Ment. Dis.*, 1932, lxxv, 22.

The total base content of the blood plasma in manic-depressive psychoses (and in other psychoses studied for comparison) is normal. The blood chlorides in the same cases are also normal. There is an increased acid-residue in the involutional depressions—of a degree, however, which is not markedly significant. The carbon dioxide content of the plasma, in the psychoses studied, lies within normal limits except in those cases complicated by: (1) 'new admission' factors; (2) toxic conditions; (3) physical abnormalities; (4) resistance to venepuncture.

No relationship exists between habitual motor activity and the degree of acidosis in those cases uncomplicated by resistance to venepuncture or 'new admission' factors.

R. G. G.

[126] **The blood pressure in schizophrenia**—H. FREEMAN, R. G. HOSKINS and F. H. SLEEPER. *Arch. of Neurol. and Psychiat.*, 1932, xxvii, 333.

IN 180 patients the mean systolic pressure was found to be 11·2 mm. lower than that of normal control; the diastolic pressure was 16·7 mm. lower. The pressure was found to have no correlation with height; there was no constant variation with season; the duration of hospitalization was found to have no effect; neither nutrition nor total daily output of nitrogen was found to bear a relation to the blood pressure. In these cases the blood pressure did not vary until the fifth decade.

G. de M. R.

[127] **Ephedrine, circulatory and glycæmic reactions in the psychoses.**—K. E. APPEL and H. D. PALMER. *Arch. of Neurol. and Psychiat.*, 1932, xxvii, 159.

TEN cases of manic-depressive psychosis were given ephedrine hydrochloride intravenously. The rise in blood-pressure was markedly greater than in ten cases of schizophrenia or five normal controls. In the manic-depressive group the blood-sugar curve showed an initial fall below the fasting level, followed by a rise reaching maximum at fifteen minutes and a secondary fall below the fasting level with a third rise towards the end of two hours. In the schizophrenic cases a marked rise occurred, reaching a maximum at the fifteen-minute period, which was sustained for two hours. In the normal person there was a sharp rise, a slight fall and a further rise, succeeded by a gradual fall toward the normal fasting level. The experiment seemed to indicate that the manic-depressive patient possesses a hyperirritability of the sympathetic, whereas the schizophrenic shows a relatively inert nervous system. None of the typical vagotonic reactions was found.

G. de M. R.

[128] **The dementia præcox child.**—WINIFRED RICHMOND. *Amer. Jour. Psychiat.*, 1932, xi, 1153.

THE writer's thesis is that an intensive study of children and adolescents, with the better psychological and psychiatric tools now at hand, is accumulating evidence to suggest that there are cases in which a deteriorating process, called præcox for want of a better term, is at work from early childhood. These cases are often thought to be mental defectives, but they are fairly easily differentiated from actual defectives. Their intelligence age first develops pari passu with their chronological age, but later it begins to slow down, and in adulthood they are on a lower level that the I.Q. promised in their childhood, and they remain on this level the rest of their lives. They seem to undergo a deterioration comparable to that which takes place in the organic psychoses.

C. S. R.

PSYCHOPATHOLOGY.

[129] **Catatonia in animals ; experimental studies of the effect of bulbocapnine and other drugs.**—GEORGE W. HENRY. *Amer. Jour. Psychiat.*, 1932, xi, 757.

PRACTICALLY all of the motor phenomena characteristic of human catatonia can be duplicated in the higher animals by means of injections of bulbocapnine in doses varying from 5 to 10 per cent. of the body weight. These reactions are dependent upon the degree of development of the nervous system. The most complete results are obtained in mammals, particularly the cat, the mouse, and the monkey. They are only imperfectly developed in birds and in the presence of external stimuli they tend to disappear. No distinctly catatonic reactions were obtained in the lizard, turtle, fish, frog, or salamander. The extent of their production is directly related to the degree of development of the neocortex. Catatonic phenomena appear gradually after the injection of bulbocapnine and the maximum effect is obtained in about half an hour. Similar reactions have been got from relatively large doses of cannabis indica and mescalin. Minimum doses of bulbocapnine produce drowsiness, reduction of motor activity, negativism and catalepsy. With moderate doses the catatonia is most fully developed. Still larger doses are followed by hyperexcitability and hyperkinesis which may terminate in tonic spasms, convulsions and death. These experiments are presented as additional evidence of toxic factors in human catatonia. The reproduction of catatonic phenomena experimentally suggests that toxic as well as psychogenic factors must be investigated before the secrets of this condition can be known.

C. S. R.

[130] **Some psychological experiments on mental defectives in relation to the perceptual configurations which may underlie speech.**—R. G. GORDON and R. M. NORMAN. *Brit. Jour. Psychol.*, 1932, xxiii, 85.

THE authors conclude that paucity of imagery is very noticeable and this is related fairly closely to intellectual capacity; the higher the intelligence, on the whole, the richer the imagery, but rich imagery does occur in the lower grades. The tendency to description seems to go with higher intelligence but does not correspond to richness of imagery. This would seem indicative of a power to analyze perceptual configurations. The noticing of colour is most frequent in the higher grades but does not occur in the lower grade subjects. There is practically no abstract imagery, the images corresponding to the limited environmental stimuli to which these cases are restricted owing to their poorly developed nervous systems. There is very

little combination of ideas into any sort of composite picture, the images which are perceived being simple configurations. This seems to explain why unfamiliar spatial configurations are more difficult than simple word configurations; also that when directly spatial configurations become complex, their analysis and consequently their manipulation become difficult or impossible. The mental defective, therefore, gives an opportunity of studying the capacity for dealing with the simpler phenomenal perceptual configurations, and further experiments might throw light on how these develop from the anthropoid to the full human level.

C. S. R.

[131] **A case of microencephaly without mental defect** (Über einen Fall von Mikroencephalie ohne geistigen Defekt).—B. Hechst. *Archiv f. Psychiat.*, 1932, xcvii, 64.

Taking 900 g. as the upper limit for the weight of microencephalic brains, the author has found records of only four in which that was not exceeded and yet no mental deficiency was present ('normophrenia'—an unpleasing hybrid neologism). The case here described was that of a woman of 73, whose brain weighed, with pia, 850 g. It was well developed and well formed for its size (the circumference of the skull was 501 mm.). She was of fair education for her social class and not obviously defective.

According to the author, the brain weight of the case corresponded to that of a six to eight months' old infant. But the important point was established by study of the case, that no arrest of development had taken place as regards the internal structure of the cerebral cortex (both cyto- and myeloarchitectonic).

S. A. K. W.

[132] **Tests of perseveration: their relation to certain psychopathic conditions and to introversion.**—J. W. Pinard. *Brit. Jour. Psychol.*, 1932, xxiii, 114.

The supposed association between the melancholic state and extreme perseveration and between the maniacal state and extreme non-perseveration appears to be confirmed by this investigation. But it directly contradicts the association previously claimed between extreme perseveration and the several qualities of character which have from time to time been supposed to indicate introversion. Not only the extreme perseverator but also the extreme non-perseverator tend to lack perseverance and self-control and to be obstinate, unreliable, 'touchy,' and 'difficult.' Whereas the perseverator tends to be 'nervous,' sensitive, effeminate and sentimental, the non-perseverator tends to be inconsiderate, tactless and critical. The moderate perseverator tends to be considerate, harmonious and reflective, the moderate non-perseverator to be courageous, jovial and a 'good mixer.'

C. S. R.

[133] **The epileptic convulsions of dementia paralytica: their relations to sclerosis of the cornu Ammonis**—H. H. MERRITT. *Arch. of Neurol. and Psychiat.*, 1932, xxvii, 138.

THIRTY cases of dementia paralytica in which sclerosis of the cornu Ammonis was found were studied. Of these cases 90 per cent. had shown epileptic phenomena. In none were any noteworthy arteriosclerotic changes found in the cerebral blood-vessels, but the conclusion is drawn that sclerosis of the cornu Ammonis is of vascular origin.

G. de M. R.

[134] **The histopathology of the brain in dementia præcox** (Istopatologia dell'encefalo di dementi precoci e zolie di disintegrazione a grappolo nel triennio 1929-31).—V. M. BUSCAINO. *Riv. di pat. nerv. e ment.*, 1932, xxxix, 342.

IN a considerable percentage (more than 50) of cases of dementia præcox the cerebrospinal fluid presents alterations generally slight but significant with the ordinary methods of examination. These alterations consist especially of a slowing of the blood-fluid exchange (a diminution of the meningeal permeability and of the rate of reabsorption of the fluid) and in a slight increase of protein without lymphocytosis. This is also reflected in the behaviour of the colloidal reactions. These are much more frequent in the catatonic hebephrenic types than in the paranoid types and in chronic than in recent cases.

For the reasons brought forward in this paper these results are not in favour of the dual conception of the pathogenesis of dementia præcox (abiotrophy and inflammatory) nor of the existence in the great majority of cases of a true inflammation of the central nervous system. They accord however, with the doctrine of a slow disintegration, minute and progressive, of the cerebral parenchyma with an accumulation of waste-products in the circulating fluid.

R. G. G.

[135] **Ear, nose and throat sepsis in mental disease**.—T. A. CLARKE. *Jour. of Ment. Sci.*, 1932, lxxviiii, 705.

THE author examined nearly 800 mental cases. Of these, perhaps 2 per cent. were selected, the remainder were routine examinations conducted irrespective of the occurrence of suggestive symptoms. All types of mental disorder were included. The claim is that focal sepsis can predispose to, and exaggerate the development of, mental symptoms; infection in the ear, nose and throat is likely to be particularly potent in so doing. The incidence of such infection in mental disease is considerable (though less than has been

suggested elsewhere); in every case therefore this region should be examined. Drastic removal of septic foci, merely on diagnosis of their existence, is no more to be commended in mental patients than in other persons. But, if other methods fail, such a procedure, carried out in a large number of cases, with adequate control, may become necessary; it may afford the only incontrovertible test of the influence of septic foci in producing and increasing mental abnormality.

C. S. R.

[136] **Epileptic reactions in the negro race.**—Nolan C. Lewis and L. D. Hubbard. *Amer. Jour. Psychiat.*, 1932, xi, 647.

The usual difficulty in obtaining reliable family histories was encountered, but in 85 out of the 160 cases there had been combinations of epilepsy, major mental disorders, excessive alcoholism, and mental defect in the near relatives, usually in the parents or siblings. In 36 family histories there had been epilepsy in the grandparents or siblings. In 36 patients there was a history of excessive alcoholism previous to the onset of the epilepsy or during its course or both. The much mooted ' epileptic personality ' was alleged to have been present in the preconvulsive lives of 16 of the 160 patients (10 per cent.). In 37 instances the attacks started in infancy and in 15 of the women the earliest seizures were associated with menstruation and the sexual epochs. As regards mental symptoms, 70 cases had outstanding confusion, and sadism was seen principally in this group. Extreme religiosity with mystical ideas was present in 19 instances and paranoid signs in 25. Actual depressive content was seen in only nine cases, which is in keeping with the low rate of depressions among the negroes as a race. Neurosomatic deterioration syndromes as evidence of disorder of the nervous system were demonstrated in 43 cases. Forty-six of the 160 were discharged from the hospital, 24 as improved and 22 as unimproved. Of the 114 remaining patients, 77 have died, 33 with status epilepticus, and the rest with asphyxiation, tuberculosis, pneumonia, cardiac failure, etc. Perhaps the most important feature noted after death was the microscopical brain picture described by Spielmeyer, consisting of a moderate diffuse cerebral neurogliosis with particular focal involvement of the hippocampus, and in addition cicatricial infarcts, minute hæmorrhages into the tissues, increase in rod-cells and neurone degenerations.

C. S. R.

PROGNOSIS AND TREATMENT.

[137] **Prolonged narcosis as therapy in the psychoses.**—Harold D. Palmer and Alfred L. Paine. *Amer. Jour. Psychiat.*, 1932, xii, 143.

The authors conclude that sodium amytal is relatively non-toxic and offers a wide margin of safety. In their investigation no patient subjected to the

narcosis treatment was made worse, while 80·8 per cent. were improved or recovered. Results showed 38·5 per cent. of complete recoveries; 42·3 per cent. of cases that were definitely improved; and only 19·2 per cent. that remained unchanged. All cases of acute mania made recoveries. The results indicated that the greatest success with this form of treatment can be anticipated in the manic-depressive group. This is directly opposed to the findings of several investigators who regard sodium amytal as a specific therapeutic agent in schizophrenia and a drug distinctly contraindicated in manic-depressive psychosis. Prolonged narcosis as a therapeutic procedure in psychiatry should be carefully evaluated by systematic trial in a large series of cases of all types. The mechanism of recovery or improvement seems to be psychological rather than biochemical. A psychodynamic formulation seems not only more reasonable and more tenable but is readily correlated with and supported by well founded psychological and psychiatric observations.

C. S. R.

[138] **Scope of psychotherapy in schizophrenia.**—Paul Schilder. *Amer. Jour. Psychiat.*, 1932, xi, 1181.

Schizophrenia can certainly be considered as an organic disease but we must not believe that an organic disease cannot be provoked by psychical causes and cannot be influenced in a psychical way. Psychoanalytic investigations so far have failed to show what the fixating events and facts in schizophrenia are, but we are justified in asking about the general problems concerned in the psychical treatment of organic conditions. These are discussed. Since a sharp borderline between organic and psychogenic must not be postulated, it is clear that as long as we do not have any reliable somatic therapy we should use our psychotherapeutic procedures. In some cases psychoanalytic approach may help but where this is not possible we can help the individual to adapt. Occupational therapy, fitting surroundings, etc., will be of importance here. It will also be possible to remove factors which provoke excitement and therefore pull the individual back strongly from reality. It is possible that even those measures will influence the process of the disease. The psychic influence may also change the appearance of the organic change. Psychotherapy is obligatory in every case of schizophrenia. The general principles involved influencing organic changes have to be applied in a careful way to the specific problems of schizophrenia. There is no question that one can do much for the schizophrenic patient but it cannot be decided yet whether we cure the schizophrenia or whether we help him to a better adaptation to reality and to his life problems.

C. S. R.

[139] **Prophylaxis in crime and delinquency of childhood.**—R. DE SAUSSURE. *L'hygiène ment.*, 1931, xxvi, 14.

THE asocial child has lost faith in the kindness of its teachers, when hatred and rebellion follow. The child must win the confidence of his teachers by realizing that he is in search of affection. Asking a child why he does this or that is to antagonize him. He realizes that the investigator cannot understand his difficulties and his lack of affection for which he craves. If the investigator gives the latter the question need never be put. In simple cases, the mere separation of child and parent for a period will cure unsociable tendencies. In cases of open rebellion, the child is frequently disarmed by the investigator making himself an ally. In the case of prison treatment, it is difficult for the doctor to appear in the rôle of sympathetic ally. It is useless being impatient with the young delinquent's lies and hostility. Encouragement alone helps to break down the initial barrier, after which the subject discloses his past and will even criticize his own reactions, however hostile, and thus understand them. For this investigation women are necessary for juveniles under 18. There is of course danger of excessive attachment to an investigator whose confidence is obtained. He wishes to be treated better than others. At this stage the doctor must behave like an 'ordinary parent.'

Above all the purely personal attitude of the child must be made clear, particularly the way in which he feels that society has wounded him.

E. M.

[140] **Insulin and nonspecific therapy: A common mechanism.**—J. I. MARGOLIS. *Jour. Nerv. Ment. Dis.*, 1931, lxxiv, 278.

AFTER the use of nonspecific therapy (including insulin) there is frequently produced a stereotyped bodily reaction—'the nonspecific syndrome,' with its two phases. The 'sympathetic phase' contains clinical and chemical findings suggestive of katabolism (increased mental and physical activities, an increase of total nitrogen and possibly pentose in the urine), whereas the succeeding 'parasympathetic phase' is one of anabolism (decreased activity, and a decrease of total nitrogen and pentose in the urine). The power to irritate the autonomic nervous system is probably present in all agents, including the specific ones (insulin, adrenalin, thyroid and so on); and the results produced are possibly due to direct stimulation of the centres controlling both antagonistic branches. This latter assumption may help to explain the tolerance and resistance to nonspecific therapy, and the apparent widespread pharmacological and possible clinical application of the Weber-Fechner law. Whether pure hormones (as crystalline insulin) are capable of stimulating only one of the antagonists (with the production of only the 'parasympathetic phase') is questionable, and further work is required to

settle this point. Thus, the ' protoplasmic activation ' after nonspecific therapy (Weichardt) takes place, but probably within the limitations of the activities of the autonomic nervous system and possibly only in the ' sympathetic phase,' for in the succeeding period there appears a marked depression of katabolism with the institution of anabolism. The diphasic character of the chemical reactions may explain some of the present contradictory chemical reports in the literature after insulin and other nonspecific injections. The autonomic nervous system does not seem to create any new defensive agents (hormones, immune bodies, and so on), but merely augments, depresses, and coordinates perhaps all mental and bodily functions, thereby producing on irritation a unified somatic response. However, at the present time, there is insufficient proof that all systems of organs in the body (skeletal muscles, skin, and so on) are directly innervated by the autonomic nervous system. This, therefore, restricts the present use of the ' nonspecific syndrome ' and its phases to a working clinical and chemical classification. Nonspecific therapy may be helpful in determining the function of any organ (with autonomic innervation) in which some obscure disease is present. It may also be of some help in the objective study of the mood and the psyche. The chemical studies (the total nitrogen and pentose in twelve-hour night urines) may be useful as a control of the clinical findings and as a guide to therapy. R. G. G.

[141] **The treatment of involutional melancholia with ovarian hormone.**—KARL M. BOWMAN and LAURETTA BENDER. *Amer. Jour. Psychiat.*, 1932, xi, 867.

SEVEN cases of involutional melancholia were treated with ovarian hormone in the form of Squibb's amniotin. The method and amount of treatment varied. Two cases showed a good social recovery; three cases were unimproved and now show a chronic picture; and two patients died of intercurrent disease. The treatment, therefore, appears to have had little or no effect on the final outcome of these cases. In giving amniotin, restoration of the menstrual cycle was hoped for, but the results were quite different.
C. S. R.

[142] **The emergency treatment of the alcoholic.**—I. D. WILLIAMS. *Jour. Nerv. Ment. Dis.*, 1931, lxxiv, 161.

THE author describes the various forms of alcoholism and stresses the point that the alcohol is used as a means of escape from unpleasant (though usually unconscious) situations. He does not believe in complete withdrawal and thinks that small quantities of alcohol are best administered neat or with small amounts of water. His routine treatment is catharsis to reduce the water content of the body and especially of the brain, followed by belladonna and hyoscyamus to counteract the overacting vagus. R. G. G.

[143] **A comparative study of sedatives employed in psychoneuroses.**—B. P. Weiss. *Arch. of Neurol. and Psychiat.*, 1932, xxvii, 956.

The author has studied the action of allyl-isopropyl-acetyl-carbamide as a sedative. He states that it acts in doses far below those that could be considered toxic and does not affect the circulation, blood-pressure or regulation of body temperature and exerts no harmful effect on respiration. It is rapidly eliminated, no trace being found in the urine on the second day. There is no change in the quantity of urine and no albumin, sugar or salt appears as a consequence. The sedative's effect is particularly rapid and this can be maintained for a longer period before sleep than is the case with barbituric acid hypnotics. The drug is stated to be of great value where a bromide is insufficient and barbiturate is too powerful.

G. de M. R.

Reviews and Notices of Books.

Integrative Psychology. A Study of Unit Response. By WILLIAM M. MARSTON, C. DALY KING, and ELIZABETH H. MARSTON. London: Kegan Paul & Co. 1932. Pp. 558. Price 15s.

THE authors have formulated their own complete system of psychology to a description of which this work is devoted. They find the dogmatic narrowness of behaviourism unsatisfactory, but, as is common in American psychological circles today, they regard investigation by introspective methods as unscientific. Their system may be regarded as an attempt at a compromise. Many of the subjects of the introspectionist, e.g., consciousness, are accepted for study but the authors employ as far as possible the objective method for investigating them.

They reject purely reflex theories of behaviour because they consider that much of the organism's activity is due to internal positive sources of energy striving to burst free. Their basic hypothesis is that the organism reacts to environmental stimuli as a whole and that this unitary response is made possible by the universal anastomoses produced by the synaptic junctions in the central nervous system. Great emphasis is placed upon the synapses, the united activity of which is regarded as the source of all mental integration.

The perception of stimuli results in four fundamental types of unitary response grouped as follows: (1) opposition and increase of activity; (2) opposition and decrease of activity; (3) alliance and increase of activity; (4) alliance and decrease of activity. These are named respectively as follows: (1) dominance; (2) compliance; (3) submission; (4) inducement. Most of the book is devoted to evolving a theory of mental life based on these four types of reaction to stimuli.

The book makes interesting reading not only on account of the authors' argument but also because of the large amount of useful, ingenious, and, let it be said, sometimes amusing experimental work that it describes. The defect of much of the literature of psychology is present, namely, the dogmatic statement of theories, formed by a priori speculation, in the vain hope of dispelling all the unknown in psychology in one volume.

T. R. H.

The Vegetative Nervous System. Edited by the Association for Research in Nervous and Mental Disease. Baltimore: The Williams and Wilkins Company. 1930. Pp. 832. Price 45s. net.

This, the ninth volume of the series edited by the American A.R.N.M.D., is both larger and fuller of fresh material than its predecessors, and forms a valuable contribution to the literature of the sympathetic system. Its thirty-five chapters are divided into five main sections dealing with morphology, physiology, recent experiment, clinical investigation, and therapy. In the earlier sections there is much new matter on the relationship of the hypothalamic nuclei to the sympathetic system. Bard's anatomical studies, and the experimental work of Beattie, Brow and Long are noteworthy contributions to this subject. In the sections dealing with recent experimental work there is new material which it is impossible to summarize. But special interest attaches to the chapter by Moore in which he shows that it is possible to remove the sympathetic ganglia completely and the chromaffin system almost completely without producing any gross pathological change in the animal. Ranson's exhaustive experiments on the relation of the dorsal roots to muscle tone are also of special interest in that they controvert to a considerable extent the teaching of Sherrington on muscle tonus.

There is comparatively little fresh material on the clinical side, if we except a suggestive paper by Duke on heat and cold sensitiveness; but the articles by Brown and Adson, Kerr and White in the therapeutic section give a very satisfactory summary of the results so far obtained in various affections by sympathectomy and paravertebral block of the sympathetic chain.

The book in fact contains much new and suggestive material which no neurologist or psychiatrist can afford to ignore. For many of his problems are elucidated by recent researches on the vegetative nervous system; and while the application of the results obtained from these to the relief of human disease is still in the experimental stage, the possibilities of advance in this direction are immense.

J. G. G.

Cytology and Cellular Pathology of the Nervous System. Edited by Wilder Penfield. In 3 vols. New York: Paul B. Hoeber Inc. 1932. Pp. 1,267. Price $30.

In his foreword the editor says that these volumes 'should be considered to some extent as the neurological representative of the series of books on cytology published by Hoeber.' But they go beyond the scope of that series for they deal not only with normal but also with pathological cytology, and even with broader subjects such as the structure of the pineal and pituitary

bodies and the retina. The third volume indeed almost leaves the subject of cytology aside, to discuss the various forms of tumour, maldevelopment, and inflammatory reaction to which the nervous system is subject; but even in this volume the subject is dealt with largely from the narrower standpoint of cellular structure. Thus the volumes cannot be considered as an exhaustive treatise on neuropathology. Indeed they make no pretence to this. But their value to the neuropathologist is scarcely lessened thereby, as they provide a foundation for his investigations such as has never before been included in one book. Many of the chapters are written by acknowledged authorities and embody their own pioneer work. Continental workers such as Agduhr, Bielschowsky, Boeke, del Rio Hortega, Ariens Kappers, Masson and Nageotte discuss subjects which they have made their own, but many of the articles by American authors are on an equally high plane.

The value of the book is greatly enhanced by the wealth of fine illustrations. To this especially must be attributed the high cost, which we regard as unfortunate, in that this is a book for slow and careful reading rather than for cursory reference in a library. But works on cytology have a permanent value, dealing as they do with carefully observed and well-established facts, and the present work contains so much fresh material that its position is assured for many years to come.

J. G. G.

Neurobiologie de l'Hallucination. By RAOUL MOURGUE. With a letter-preface by HENRI BERGSON. Brussels: Maurice Lamertin. 1932. Pp. 416. Price 91 fr.

DR. R. MOURGUE has been occupied for a number of years with problems circling round and arising out of the question of hallucinosis, and this erudite and stimulating book embodies the results of his studies. Convinced that academic psychology will never provide a solution of the nature of hallucinosis, Dr. Mourgue asserts that, in the first place, the latter does not depend on the intensity of the image phenomena, nor on disaggregation of personality in any simple sense; he has found, however, that in abnormal perception (hallucination) much depends on complex motor attitudes, through which objectivity is determined by reference to the body. Normal perception is based on external sensory excitation; hallucination is based on motor attitudes that have to do with projection—it is a ' psychosomatic ' phenomenon. Dr. Mourgue then proceeds to argue that true hallucinations originate in disorder of the organo-vegetative side of the personality; this system is the ' base physique de l'esprit.' Along with the organo-vegetative factor goes activity on the part of the corresponding motor mechanisms of sensorial adaptation. Such interpretations of the phenomena, however, are

almost entirely physiological; the author is therefore led to insist also on the significance of the sphere of orientation and causality, which, in his view, is invaded by that of instinct when hallucinations arise. Hallucination depends on activity in the instinctive sphere, which uses for its own purpose the spatial projection apparatus normally serving for orientation in the world of reality.

This is a mere sketch of the general trend of Dr. Mourgue's investigations; the book itself offers a closely reasoned and well sustained argument in favour of these views, and is remarkable for its range of knowledge in presentday research. He touches not merely on anatomical and physiological data, but handles purely philosophical considerations with equal zest. A bibliography of over 1,000 titles indicates the breadth of Dr. Mourgue's acquaintance with the germane literature.

L'Épilepsie chez l'Enfant et le Caractère Épileptoide. By GILBERT ROBIN. Paris: G. Doin et Cie. 1932. Pp. 150. Price 18 fr.

THE writer of this attractive little book is imbued with the idea that an 'epileptoid constitution' exists, and can be recognized. He believes that it manifests itself by traits and symptoms such as volatility, instability, bad temper, impulsiveness, sometimes violence, cruelty, nocturnal incontinence, somnambulism, and what not; and then seeks to persuade his readers that such a 'character' is traceable in youthful persons who have never suffered from overt fits. In other words, he is convinced of the genuineness of 'épilepsie sans crise.' A large number of brief clinical case-reports will be found in these pages, whose interest is unquestioned. Nevertheless the impression is left after perusal that too much is assumed, and that the writer is not guiltless of a tendency to petitio principii. For, after all, we are not here vouchsafed a definition of epilepsy, nor are its limits prescribed. Every clinician knows that confirmed epileptics may exhibit 'mental' symptoms; less recognition has been given to the equally well substantiated fact that many persons who suffer from occasional fits do not show such symptoms. Yet numerous authorities appear to be assured of the genuineness of the epileptic 'character' or 'mentality' or 'disposition' or whatever word may be preferred, and go so far as to affirm that fits themselves are of secondary importance from a diagnostic viewpoint. This seems to the reviewer an excellent instance of putting the cart before the horse. The truth is that under the sheltering wing of epileptic 'constitution' has been crowded one of the most heterogeneous collections of mental abnormalities, from religiosity to mythomania, and from brutality to humility, that can be imagined. Thus packed with anomalies, the conception becomes meaningless.

Humoral Agents in Nervous Activity. By G. H. PARKER, Harvard University. Cambridge: University Press. 1932. Pp. 79. Price 6s.

THOUGH this monograph deals with a special aspect of comparative physiology, viz., the problem of chromatophoric activity in lowly species, it has implications of great importance for neurology in general. Professor Parker first shows that chromatophores are influenced by neural action in some animals, by humoral in others. Colour cells are responsive both to neural stimuli and to adrenalin, pituitrin, and other substances of a humoral class; that is, the cells have a dual control. He next observes that nervous operations excite secretory processes and theorises on the possibility that nervous tissue itself is endowed with secretory function. Evidence is advanced which goes to prove that some secretions appear to come from the nerve-terminals of sympathetic fibres, that some or other substance in the nature of a hormone may be given out by fibrils—e.g., in the case of taste-buds. Thus the question comes to be one of distinction between normal (sensory) nerve impulse and trophic influence passing over the same nerve-fibre to keep its sense-cells intact; the latter must pass in a reverse stream from centre to periphery. The suggestion is made that herpes zoster supplies an instance in support of the neurohumoral principle; the inflamed skin over the segmental distribution may be due to abnormal secretions from the nerve-terminals of the skin secondary to the deeper (ganglionic) nerve disturbance. Finally, the notion is used to account for irreversibility at synapses; facts are adduced in support of the view that it has a secretory and a receptive side, and that these are structurally different. Professor Parker concludes that the principle is a special application of Bayliss and Starling's concept of hormones, though the distance between origin and point of application of the substance may be microscopical.

Selected Writings of John Hughlings Jackson. Vol. II. Edited by JAMES TAYLOR, M.D., F.R.C.P. London: Hodder and Stoughton. 1932. Pp. 510. Price 25s.

THE second volume of Hughlings Jackson's contributions to neurological science includes a number of papers dealing with one of his favourite topics, viz., evolution and dissolution in the nervous system, with aphasia, and also sundry addresses and lectures. The editor has selected with care and exercised a wise judgement; among the miscellaneous articles are several which reveal Jackson's mind at least as effectively and characteristically as some that are much more ambitious. His little jeu d'esprit on the Psychology of Joking is as delightful in conception as it is informative in its application to 'more serious' matters. His brief remarks on non-protrusion of the tongue in some cases of aphasia contain in essence the idea

of the doctrine of apraxia as known today. The first Hughlings Jackson Lecture, delivered by himself in 1897, is remarkable in more than one respect for its modern outlook as regards epilepsy. It is an intellectual treat to peruse these diversified pages at leisure, and, for those who knew the man, to recapture some echoes of his voice, some of his charm, and to be impressed again by his wonderful insight into the heart of the problems that attracted him. Those who did not known him will not fail to appreciate the quality of the legacy he has left and to profit by study of its richness.

Chemical Wave Transmission in Nerve. By A. V. Hill, F.R.S. Cambridge: University Press. 1932. Pp. 74. Price 5s.

Extremely technical though this little book is, it is equally fascinating; its distinguished author begins in the shallows and leads the reader into ever deeper waters in such a manner that the latter hardly realizes when he is out of his depth. The ' messages ' of nerve-currents passing along nerves cannot be discrete packets of any actual substance. Nor is there any evidence that the nerve impulse is a special form of energy. There is no such special thing as ' nervous energy,' despite the frequency with which the term is employed. The nerve impulse is an event. It is transmitted along a protoplasmic thread like an electric cable with conducting core and insulating cover. With it goes an electric change that seems to be a universal accompaniment; this change can be used as a sign of the presence of the impulse and as a measure of its size. In addition, heat formation, oxygen consumption, and carbon dioxide production occur at the same time. The magnitude of the impulse cannot be varied (' all-or-none ' principle); and its passage is followed by a refractory period; as Prof. Hill says, ' a gun has been fired—nothing can happen till it is reloaded.' The different fibres of a given nerve-trunk have different diameters, and the velocities with which impulses travel are proportional to these diameters.

According to the author, the problem of impulse transmission is precisely that of electrical excitation; a nerve fibre is to be regarded as a cylindrical condenser. He then proceeds to examine in detail the analogies and the differences. When currents flow, ions must move. The phase of recovery can be likened to a secretary process, which ' forces back to their place ions which have escaped during activity, ions whose diffusion has caused the electric currents which we observed.' Recharging and secretion are comparable. How this secretion is done, ' nobody knows, and nobody is likely to know for some little time.' Yet something of the kind must occur, for when the resting potential of nerve is diminished by stimulation (with its consumption of oxygen), rest in oxygen raises it again,

Biologische und reine Psychologie im Persönlichkeitsaufbau. By Prof. Dr. G. EWALD, Erlangen. Berlin: S. Karger. 1932. Pp. 138. Price Mk. 14·40.

PROF. EWALD in his preface explains the purpose of his monograph. He thinks it time the psychiatrist should again turn to examination of the somatic components in personality structure. Not that he wishes to break a lance on behalf of the 'old materialistic soul'; but since we can only know the mind as it exists in connexion with and relation to the body he considers psychical processes, normal and abnormal alike, should be envisaged also from the standpoint of body structure. In this there is, needless to say, nothing new, but it serves a useful purpose to investigate afresh the biological side of temperament and character. This is done in a series of pleasantly written chapters, the views of several contemporary theorists being discussed and the 'make-up' of hysteroid, schizoid, and 'demonic' types being examined. One is devoted to individual psychology, another to the place occupied in character-formation by the sexual impulse, as conceived by Freud. Prof. Ewald considers Freudian doctrine makes but a minor contribution to characterology, being of more value for psychopathology. From Freud little is to be learned of normal character structure. 'Like a colour-filter, which only allows red rays to pass,' so Freudian concepts never convey the idea of the personality as a whole, but only of one of its aspects. Necessary and desirable though this has been, Freud 'filters only the sexual through,' and deals with it alone; thus his conception of personality is forced.

Le Système Nerveux de la Vie Végétative. By D. DANIELOPOLU. (La Pratique Médicale Illustrée, edited by E. SERGENT, R. MIGNET, and R. TURPIN.) Paris: G. Doin et Cie. 1932. Fasc. I: pp. 100; price 30 fr. Fasc. II: pp. 110; price 30 fr.

PROFESSOR DANIELOPOLU of Bucarest is the author of this comprehensive treatise on the sympathetic nervous system. Its first volume is devoted to normal anatomy and physiology, the second to clinical examination, pathology, and medico-surgical treatment. Well illustrated with original diagrams, many of which are coloured, it is particularly detailed on the physiological side. Three fundamental principles are enunciated in this regard, viz., the amphotropism of sympathetic tonus, predominance of amphotropic factors on one or other group (sympathetic or parasympathetic), and circular amphotropic mechanism of tonus and excitability. These notions are applied to the clinical investigation of sympathetic disorder in an ingenious fashion. Attention is also engaged by a series of syndromes in which sympathetic disturbance is manifest (asthma, angina, hypertension, tabetic crises, and many others). The book is based on personal experience and research, and represents a high level of attainment; but there are no references to the work of others, although their names are occasionally quoted.

Report of the Departmental Committee on Persistent Offenders. Presented to Parliament, May, 1932. London: H.M. Stationery Office. 1932. Price 1s. 6d.

Of the eight persons constituting the above-mentioned Departmental Committee only one was a member of the medical profession. They have sifted with care an immense amount of evidence derived from heterogeneous sources, part of which interests the neurologist and psychopathologist. Various witnesses testified to the importance of mental examination of persistent offenders, juvenile or adult, and some averred that their criminal habits could be cured by appropriate psychological treatment. They alleged that punitive treatment is as little helpful as it would be for a sufferer from any recognized medical disease. Such statements are based on the assumption that many crimes are merely symptoms of mental disorder. It is of value, therefore, to possess the Committee's conclusions, couched in temperate language, and certainly not biased one way or the other.

In the first place, they do not agree with the view that crime is a disease, or that it is generally the result of mental disorder. They think, however, that a certain amount of persistent crime is due to abnormal mental factors, and that certain delinquents may be amenable to psychological treatment; yet, accepting the psychological explanation in certain circumstances, they do not regard it as of necessity an excuse for the offence. The Committee believes that some reduction in the amount of certain forms of persistent crime might be brought about if more use were made of chances of examination of various offenders at reputable clinics. They are unwilling to express any definite opinion as to the relative value of psychological treatment prior to, or during the currency of, imprisonment.

With a final conclusion we find ourselves in cordial agreement; it deserves to be quoted verbatim:—'We should be on our guard lest words are substituted for facts until the efficacy of treatment is based on scientific data. Meanwhile, we consider that action along these lines would determine whether the psychological treatment of delinquency is of sufficient value to justify statutory recognition as a means for the prevention and treatment of certain crimes.'

Human Sterilization. By J. H. Landman. New York: The Macmillan Company. 1932. Pp. 341. Price $4.

Dr. Landman's book contains no propaganda; it presents a dispassionate and scholarly discussion of the question of sterilization, based on experience in the United States. At the time of writing, the author finds that 27 States of the Union have passed legislation bearing on the matter, and that some 12,000 persons have already been sterilized. With his general

attitude all medical scientists must agree, that the crux of the problem is the question of defective inheritance, and that what is sorely needed is more data and less speculation. His study is particularly well documented; all the known facts are collected and analyzed; the results of sterilization, so far as available, are here marshalled and made to run the gauntlet of criticism. Since biologists and eugenists are still very much in the dark as regards transmissibility of characters it is, to say the least, premature to regard the problems as solved; but without this solution, the procedure becomes crassly empirical. Dr. Landman shows that many mental incompetents, who have been submitted to the operation (whichever one is chosen) have none the less to be returned to institutional life; if they need such care, why sterilize them? He also shows that some inmates respond to treatment and eventually become stable and well-conducted. If they can be thus discharged, why sterilize them?

Publicists whose knowledge of the subject is too often amateur might with advantage read the evidence so clearly and concisely set forth in this volume.

Les Séquelles de l'Encéphalite Épidémique. By GEORGES GUILLAIN and PIERRE MOLLARET. (La Pratique Médicale Illustrée, edited by E. SERGENT, R. MIGNOT, and R. TURPIN.) Paris: G. Doin et Cie. 1932. Pp. 104. Price 28 fr.

THE major part of this well compiled monograph deals exhaustively with the sequelæ of epidemic encephalitis and ranges over an enormous field, from amyotrophy to psychosis, from Parkinsonism to arthropathy. All the numerous sections and subsections are provided with bibliographical references which will prove of value to anyone studying the subject further. Brief chapters on etiology, diagnosis and treatment are included. The tone of the book, as befits its purpose, is descriptive rather than critical or interpretative, although on various matters in dispute the writers express an opinion of a constructive kind. It is an erudite production which we cordially recommend to the notice of our readers.

Das Rechtsgefühl in Justiz und Politik. By Prof. Dr. A. E. HOCHE, Freiburg i. Br. Berlin: Julius Springer. 1932. Pp. 102. Price Mk. 5·80.

PROF. HOCHE has written a thoughtful and informative essay on the sense of right, the feeling of justice and injustice, exemplifying the shades of meaning attached thereto. He gives an entertaining account of how one's sense of what is just or right may be trampled on in a railway carriage, at a hotel, a concert. His illustrations, indeed, are peculiarly apt; another cameo

depicts the injustice of unequal division of food at the nursery table and how it is received; still another is based on *l'affaire Dreyfus* as an instance of outrage to this sense or feeling. Prof. Hoche shows that in abnormal circumstances the same feeling bears on the development of martyrdom, of resignation, and how it is well to the fore in the class of querulants. That the feeling is engrained in mankind is affirmed, yet not so deeply or so universally as could be wished—nothing like it, perhaps; it is but the first and most delicate bud, as he says, the harbinger of a summer when no one will be compelled to do this or leave that, but everything will be done for the common good not by compulsion but of choice. A Utopian dream, no doubt, yet possibly not without significance.

Le Traitement Actuel de la Paralysie Générale. By Dr. Pierre Mollaret, Paris. Paris: J. B. Baillière et Fils. 1933. Pp. 68. Price 8 fr.

This slender brochure contains a very complete and succinct description of all the newer methods of treatment of general paralysis in vogue today; half is devoted to the details of 'pyretotherapy' and chemiotherapy, the rest of the book to results, recommendations, criticisms, prophylaxis and symptomatic treatment. In convenient form with numerous references, the reader will find here all that is necessary for guidance from the standpoint of practice.

Les Fonctions Sexuelles Mâles et leurs Troubles. By Stanislas Higier. With a preface by Prof. Laignel-Lavastine. Paris: G. Doin et Cie. 1932. Pp. 391. Price 50 fr.

Dr. Higier's study of the sexual functions of the male and their disorders is intended to serve merely as an introduction to a further and more elaborate investigation of impotence; as it is, however, it deals with the subject from the standpoint of physiology and endocrinology at least as much as from that of psychology, for which he is to be congratulated. These aspects of an important clinical syndrome have been badly neglected of late, since the permeation of current thought by Freudian conceptions has too often circumscribed research instead of widening it. At one end of the scale of impotence come cases where loss of sexual power depends on disease of the spinal cord, roots, or nerves; at the other, the subjects of phobia, hypochondria and one or other psychoneurosis, neurosis, or psychosis, in whom the symptom or syndrome is doubtless psychogenic. But between these, as Dr. Higier is careful to point out, is a large number of cases in which the origin of the symptoms in disorder of vagosympathetic or endocrine mechanisms seems clear. To these much attention is devoted. The book is eminently practical, well written, and possesses that rarity of French medical literature, an index.

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Experimenteel anatomisch Onderzoek omtrent het Central Acustische Stelsel. By F. E. Posthumus Meyjes. Amsterdam: Druk. M. J. Portielje. 1932. Pp. 107.

Following lines of study already investigated by his *maitre*, Prof. B. Brouwer, Dr. Meyjes has devoted his thesis to the question whether there is any topical connexion between the internal (median) geniculate body and the temporal (auditory) cortex, and whether any corticifugal system exists from the latter to the former—i.e. whether there are efferent fibres in an afferent system, as has been shown to be the case in respect of the visual system. The answer to the first problem is that geniculo-cortical relationships are diffuse rather than precise; there does not appear to be in the internal geniculate that subdivision of ganglion-cells which characterizes the external. On the other hand, however, a well marked efferent tract can be seen to pass from the temporal lobe (auditory cortex) to the internal geniculate. Dr. Meyjes properly attaches much significance to this path, suggesting that its physiological influence is exerted on the lower sense station by way of a possible combined excitation and inhibition; those parts which are placed in the centre of observation become more susceptible to stimuli, others less; in a sense, therefore, the system is one for focussing attention. Dr. Meyjes' work is bound to prove fruitful as a contribution to a subject that will attract increasing notice.

Fleckfieber und Nervensystem. By Dr. Nikolai Hirschberg, Moscow. Berlin: S. Karger. 1932. Pp. 124. Price Mk. 11·60.

For clinical and pathological experience of typhus English neurology has to depend today on that of foreign observers in countries in which the disease is still rife. This monograph from Moscow presents a complete picture of the nervous aspects of the fever, which are extraordinarily wide-ranging. We are informed that the symptom-complexes include myositis and neuritis on the one hand, and psychoses on the other. Spinal, bulbar, cerebellar, pyramidal and extrapyramidal syndromes are all met with—in a word, any whose basis in an encephalo-myelo-neuritis can be assumed. On the pathological side full descriptions are given of the histology of the well-known lesions, unspecific though they have been shown to be. The bibliography contains nearly three hundred titles.

The Diagnosis of Nervous Diseases. By Sir James Purves-Stewart, K.C.M.G., C.B., M.D., F.R.C.P. Seventh edition, revised. London: Edward Arnold and Co. 1932. Pp. 730. 35s.

The seventh edition of this well known and popular textbook has been revised with the introduction of new matter in the chapters on anatomy and physiology, in an account of postural reflexes and in the discussions of

intracranial tumours and disorders of sleep. It will continue to fill a want which is supplied by no other book in the English language. It is a pity, however, that certain theories are given prominence as though they were really proved explanations, e.g. the sympathetic innervation of striated muscle. Exception too may be taken to such a dogmatic statement as that optic atrophy does not occur in Friedreich's ataxia. As in previous editions the book is lavishly illustrated and there is a good index, both features which add to its value.

Experimental Catalepsy. By ARMANDO FERRARO and S. S. BARRERA, New York State Psychiatric Institute and Hospital. Utica, N.Y.: State Hospitals Press. 1932. Pp. 119. Price not stated.

THIS monograph provides the reader not only with a complete mise-au-point of the present position of the catatonia problem but also supplies many valuable data of personal experimentation in connexion therewith. The authors have employed monkeys and cats, and come inter alia to the following conclusions.

The cataleptic phenomena producible by bulbocapnine include loss of motor initiative, maintenance of passively impressed postures, maintenance of posture against gravity, resistance to passive movements, and variations of muscle tonus. This bulbocapnine catalepsy can be induced in cats and monkeys from which part or the whole of the cortex has been removed. These facts of experimentation confute the claims of those who believe that the cortex is essential for the manifestations.

It is significant to note that the authors affirm that this experimental catalepsy is not identical with the catatonia of human cases—it represents only its motor component, and does not embody any psychical component. Whether this conclusion is just is, we consider, open to discussion. In the psychoses catatonia is declared to be psychologically determined; in the ape, the clinical phenomena are exactly the same except for evidence of psychical participation. It is legitimate to argue that one and the same mechanism is activated in different ways in the two conditions, just as the plantar response may be absent when the foot is cold, under certain abnormal spinal cord states, and in hysteria.

The Biological Tragedy of Woman. By A. NEMILOV. Translated by STEPHANIE OFENTAL. London: G. Allen & Unwin. 1932. Pp. 220. Price 7s. 6d.

THE medical man will not learn anything new from this book, but none the less it is interesting in respect of the different point of view which a Bolshevik protagonist must hold when compared with an exponent of western thought.

The book is written on the theme that a woman is a human animal who menstruates once a month and may bring forth once a year.

According to the Soviet régime it is axiomatic that woman stands on an absolutely equal footing with man, but the author points out in a popular but clear and accurate description of sexual physiology in the female from birth through puberty, menstrual function, pregnancy, lactation and the menopause, that she cannot compete on terms of real equality. Naturally he is an upholder of birth control, but thinks that the future lies with antispermic sera rather than with the methods at present in vogue. He protests, however, that sexuality must be controlled since it can be and is sublimated into all varieties of culture and art. He mentions that it is sometimes diverted into religious channels but alleges that this is always pathological.

He proposes the following as the 'new ethic of sexuality,' which, it must be confessed, bears a distinct resemblance to the old.

1. Sexual life should be valued and guarded, for it is 'life's greatest blessing.'

2. Sexual life should be regulated for purely biological reasons.

3. Each sexual union should be regarded as a responsible act from the point of view of the interests of human society.

4. In arranging sex life due regard should be given to what has been described as the biological tragedy of woman.

Evidently his views are not acceptable to some of his compatriots since he finds it necessary to defend himself in an epilogue against charges of an un-Marxian attitude.

R. G. G.

Nursing Psychological Patients. By MARY CHADWICK, S.R.N. With a foreword by DAVID FORSYTH, M.D., F.R.C.P. London: George Allen and Unwin Ltd. 1932. Price 10s.

THE object of this book is to give to those who nurse cases of psychological disorders an explanation of the origins of their symptoms. There is no mention here of ordinary methods of nursing. In reality the work may be regarded as a short primer of modern psychological teaching for nurses. It is written in language as simple as the difficulties of the subject will allow. Even then we doubt if it will not prove too big a mouthful for many of those for whom it is intended. As an introduction to the whole subject of the psychoneuroses it can be recommended to medical students or even the laity. A bibliography at the end of the book and at the end of each chapter are useful features for the reader who would proceed further in his studies.

Persönlichkeitsentwicklung und Wahnbildung. By Dr. med. et phil. WALTER BETZENDAHL. Berlin: S. Karger. 1932. Pp. 321. Price Mk. 16.

THE object of this presentation is to study those disorders of the mind which, however diverse their manifestations, are alike in depending on changes in

the ego. Nearly the whole monograph is taken up with the descriptions of individual cases, at length and in detail, and with comments on each. In the endogenous psychoses disturbance of this 'Selbstgefühl,' this sense of self, is a central feature, but the writer deals interestingly with minor fluctuations distinguishing persons who are normal, and shows how intermediate grades are met with. He discusses the factors responsible for such variations. The book is conceived on a purely empirical plane, and no special theory is advocated; it possesses documentary value, however, and the analyses of the case-reports are instructive.

Klinisch-anatomisch Onderzoek over Neurologische Ademhalings stoornissen. By J. W. G. Ter Braak. Assen: Van Gorcum Co. 1932. Pp. 163.

This thesis from the Dutch school consists of a clinico-anatomical research on disorders of respiration produced by lesions in the central nervous system. As the neurologist is well aware, the apparatus of respiration subserves more functions than that 'vegetative' process with which the experimental physiologist is concerned. The respiratory movements of speaking and for the expression of the emotions, etc., cannot be examined in an animal laboratory; and in this respect the clinico-anatomical method can supply information which will add to knowledge. Dr. Ter Braak distinguishes the voluntary function of respiration (speaking, etc.); its emotional function (laughing and crying); and its automatic (vegetative) function. The apparatus has a cortical (pyramidal), subcortical (extrapyramidal) and bulbar innervation. He exemplifies the relationship between these by a series of carefully studied cases, and their not infrequent dissociation—as sketched by Hughlings Jackson many years ago. The thesis is well illustrated and posseses the advantage for English readers of a brief summary in that language.

Probleme der klinischen Psychiatrie. By Prof. Kurt Schneider, Munich. Leipzig: Georg Thieme. 1932. Pp. 46. Price Mk. 2·60.

This short critical essay deals with vexed questions of nosology in regard to mental disease. Confusion arises when canons of nosology applicable to disease of the body are utilized for disorders of the mind. Some mental affections are distinguished from each other by symptoms that are not mental. It is preferable to deal in syndromes, or type reactions. Prof. Schneider considers the following can be differentiated: cyclothymias, episodic psychoses, deliria, schizophrenic processes, paraphrenias, and dementias. This psychiatric arrangement is purely symptomatic; clinical psychiatry is not pathopsychology. Moreover, the author admits that the scheme cuts across natural biological relationships; thus an alcoholic

hallucinosis might be reckoned with episodic psychoses, deliria, or paraphrenias. Where etiology is unknown purely psychological labels are chosen. Mixtures are in constant vogue—e.g. *paralytic* dementia, *involution* paraphrenia, *febrile* delirium. Distinctions between types are often extremely difficult; the question of psychosis or abnormal personality is sometimes unanswerable.

Though no constructive conclusion is arrived at, this unpretentious contribution merits notice for its scientific spirit and for its frank refusal to gloss over the inconsistencies and, in some respects, the absurdities of contemporary psychiatry.

Nervous Disorders in Infancy and Childhood. By NEILL HOBHOUSE, M.D., Physician to Out-patients, Victoria Hospital for Children, etc. London: H. K. Lewis. 1933. Pp. 207. Price 8s. 6d.

DR. HOBHOUSE is the author of an unpretentious volume dealing with nervous troubles of all kinds that affect infants and young children, and manages to supply a great deal of practical information within relatively small compass. The book bears the impress of personal experience and of independence of mind, and presents its subject-matter in readable form. The chapters on undesirable habits, on neuroses and psychoneuroses, on mental arrest and deficiency, contain interesting discussions of a simple yet penetrating kind on the problems with which they are concerned. A number of comparatively rare conditions receive brief notice. In general the paragraphs on treatment are sensible and adequate. There are few references to the literature, and at one or two points we come across a tendency (somewhat prominent in these times) to simplify a subject by consideration merely of the latest paper thereon, irrespective of what has long been known and recognized. Thus scant justice is done to meningitis serosa, to which, indeed, a misleading allusion is given.

Reflex Activity of the Spinal Cord. By R. S. CREED, D. DENNY-BROWN, J. C. ECCLES, E. G. T. LIDDELL, and C. S. SHERRINGTON. Oxford: Clarendon Press. 1932. Pp. 183. Price 10s.

THE collection in one volume of a series of reviews, as they may be termed, illustrating the features of reflex mechanisms as carried out by the mammalian spinal cord, serves a highly useful purpose. Although the clinican daily resorts to reflex examination for diagnosis, or studies reflexes for other reasons, he needs to keep himself abreast of physiological advance, and this timely book provides the means whereby he may do so. Reflexes are considered in their physiological and not their biological setting; their minutiæ are studied with the greatest fullness, but since the scope of the

work, anatomically, does not extend beyond the midbrain, the reader must keep in mind the one-sided nature of the presentation here offered. On the other hand, he will learn much indeed that is of prime clinical importance, even though the authors disclaim any other viewpoint than that of pure experimental physiology.

Crises Gastriques du Tabes. By ADOLPHE HOROWITZ. Paris: G. Doin et Cie. 1932. Pp. 120. Price 25 fr.

IN a critical review of the vexed question of tabetic gastric crises and their treatment stress is laid on the inefficacity of conventional medical therapeutics; morphia and its congeners are condemned roundly; and surgical procedures are shown to be valueless in at least 60 per cent. of cases in which they have been tried. Dr. Horowitz also points out the uselessness of antisyphilitic measures for the crisis itself. He claims to have had excellent results in those of sensorimotor or sensory type from intravenous injection of neutral sulphate of atropine; the first injection contains $\frac{1}{2}$-1 mgm., the second, some hours later, $1\frac{1}{2}$-2 mgm. Since 1928 he has made over 200 injections without complication of any kind, and affirms that the method is never contraindicated except perhaps in the case of cardiac insufficiency with bradycardia.

Normal Youth and its Everyday Problems. By DOUGLAS A. THOM, M.D. New York and London: D. Appleton and Company. 1932. Pp. 383. Price 10s. 6d.

THIS book may be regarded as a sequel to the author's *Everyday Problems of the Everyday Child*. Dr. Thom's aim has been to produce a useful manual which will help those concerned with developing youth, particularly parents and teachers, to handle the various situations likely to arise during this difficult period. His long experience with children, together with his practical acquaintance with general psychiatry, has enabled him to approach the problems from a wide angle, and to put forward balanced views. The book is written throughout in simple and untechnical language, but it contains much practical wisdom, and should be of decided service to the readers to whom it is addressed.

Arbeiten aus dem Neurologischen Institute an der Wiener Universität. Vol. XXXIV. Leipzig and Vienna: Franz Deuticke. 1932. Pp. 307. Price M. 60.

THE latest volume in this well-known series contains a number of papers meriting notice. A long article on the changes in the central nervous system accompanying scarlatina and diphtheria adds to the many which have

appeared of recent years dealing with the interrelation of fevers and exanthems and nervous phenomena. It deals in considerable detail with the interesting and vexed problem of cell-inclusions and their significance. Another paper gives a good description of atrophic sclerosis of one hemisphere. A complicated case of syringomyelia, with taboparesis (metalues), and with trauma and tumour-formation as additional causative factors is narrated, and well illustrated. The pathology of a case of paralysis agitans forms another topic, showing the difficulties of ascribing the clinical symptoms to the lesions found post-mortem and exemplifying the diffuseness of the lesions in some specimens of the disease.

Intracranial Suppuration. By E. Miles Atkinson, F.R.C.S. London: Jonathan Cape. 1932. Pp. 127. Price 5s.

Mr. Atkinson as an otologist and laryngologist has had a wide experience of the intracranial complications of local sepsis in ear and paranasal sinuses, and has written a useful book, small in compass, for the general practitioner. It forms a reliable guide to practice and treatment in respect of these states, though its approach is to some extent conditioned by the special material with which it deals. The author discusses seriatim the complications involving skull, membranes, and brain; he covers the ground systematically, handling his subject also on broad lines so as to give the reader as wide a view as is practicable. The neurologist may cavil at some of the statements, and criticize Mr. Atkinson's occasional representation as something relatively new of what is in reality quite the reverse; but taken as a whole the book efficiently serves its ostensible purpose.

Handbuch der biologischen Arbeitsmethoden. Edited by Geh. Med.-Rat Prof. Dr. Emil Abderhalden. Abt. V, Teil 7, Heft 8. Berlin and Vienna: Urban and Schwarzenberg. 1932. Pp. 995-1173. Price Mk. 10.

The latest part of Prof. Abderhalden's monumental treatise, or rather encyclopædia, contains four articles dealing with the special senses. Of these, two are concerned with comparative physiology—the non-acoustic functions of the labyrinth in fishes, cyclostomes, amphibians, and reptiles. Another deals technically with the production of tones of different frequency and intensity, the last with the methodology of the sense of touch. This discusses at some length the information to be derived from palpation of different parts of the body, especially the abdomen. The refinements illustrated by the author seem in some respects almost unattainable, though his diagrams must be based on the results of such technique, and they are admittedly rather impressive.